

CHILDREN'S DISEASES

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FOREWORD

The present tenth edition of "Children's Diseases", by Professors A. Koltypin, N. Langovoi and V. Vlasov, has been revised by Professors V. Vlasov and B. Shirvindt. The objective of this textbook is to provide the medical student with basic information on first aid to sick children and the care and treatment of children in hospitals. While particular attention is given to material pertaining to clinical observation, care and medical treatment, which is presented in great detail, considerable space is also devoted to pathogenesis, as correct treatment is impossible without an understanding of the causes and mechanisms of the given disease.

Hygiene, care and food being factors of prime importance in the treatment of sick children, special attention is given to the sections of the textbook that deal with those subjects.

The tables in the appendix will, the authors believe, be of practical value to graduate nurses and pediatricians.

CHILD WELFARE, MEDICAL AND PROPHYLAXIS SERVICES FOR CHILDREN

Before the Socialist Revolution there was no state child welfare service in Russia. The existing children's institutions—children's homes and orphanages for abandoned infants—took very little interest in child health protection. Mortality in those institutions was sometimes as high as 90 per cent.

There were out-patient clinics and hospitals for sick children but far from sufficient in number, and they did nothing at all to prevent disease. The physician's job was to treat visiting sick children, and no more, in the out-patient clinics, and in the hospitals—patients whose type of disease called for hospitalisation.

In the very first months after the Revolution a Mother and Child Welfare Department was organised under the People's Commissariat for Assistance to the Destitute. A decree proclaimed in January 1918 set forth the objectives and ways of development of mother and child welfare services. This decree was the first ever to declare that mother and child welfare was a matter of state concern.

The former orphanages were reorganised into homes for babies, nursery schools were opened, and medical consultation centres, first for infants and later for children up to the age of three, were founded. The children's out-patient clinics were enlarged and their activities extended to include preventive medical services, carried out in close contact with schools and all kinds of health promotion centres for children, such as playgrounds, summer camps, and so forth.

As they developed, the children's medical centres began to set up dairy kitchens where milk formulas and other baby food was prepared and made available to children, both healthy and ill.

Existing children's hospitals were expanded and new ones opened in many towns. The number of children's institutions grew from year to year and is still growing.

The large-scale organisation of child welfare in the U.S.S.R. created an increasing demand for pediatricians. Such specialists are trained in medical schools, specialised pediatric research institutes and children's hospitals.

RUSSIAN PEDIATRICIANS

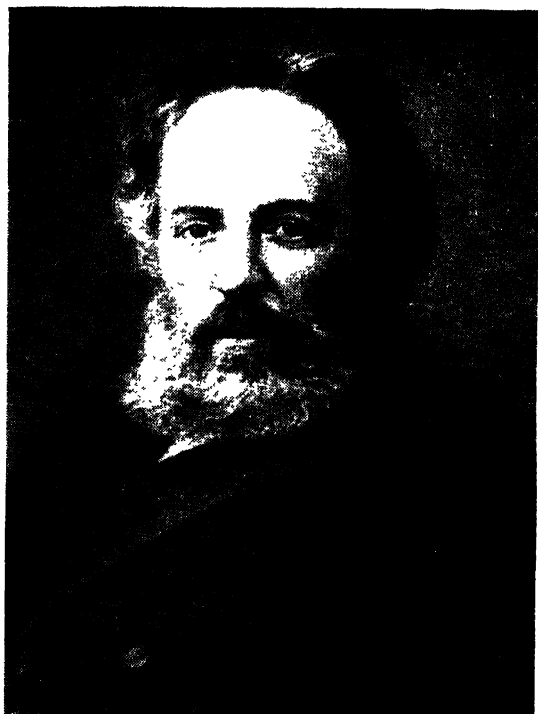
Soviet pediatrics has a notable tradition to draw on. It has developed along original lines and made a valuable contribution to world medical science.

As early as the 18th century the famous Russian scientist M. Lomonosov (1711-1765) gave much attention to the problem of child welfare. In a letter to I. Shuvalov (a dignitary during the reign of Elizabeth of Russia) he pointed out that the state should provide obstetric aid and suggested measures for combating children's diseases and infant mortality.

An important part in the development of Russian pediatrics was played by S. Khotovitsky (1796-1885), who is regarded as having



S. Khotovitsky



N. Filatov

been the first Russian pediatrician. As head of the Chair of Obstetrics, Gynecology, and Children's Diseases at the Academy of Medicine and Surgery in St. Petersburg, he was the first to teach pediatrics as a separate branch of clinical medicine. Khotovitsky was also the author of the first Russian textbook on children's diseases, *Pediatrika (Pediatrics)*.

Mention should be made of the eminent Russian pediatricians, founders of the branch of pediatrics in medical science in Russia—N. Filatov and N. Gundobin.

N. Filatov (1847-1902), professor at the children's clinic under the Moscow University, managed, despite the brevity of his life, to make a valuable contribution not only to Russian, but also to world pediatrics. His *Semeiotics and Diagnostics of Children's Diseases* was a handbook for Russian pediatricians. It was translated into many foreign languages and recognised as the best reference book of its kind.

Filatov was one of the first to describe, in 1885, the prerash (prodromal stage) symptom of measles—small red squamative

spots surrounded by white areas in the mucous membrane of the mouth; somewhat earlier this symptom was reported on by a country doctor, Belsky. In the Soviet Union this symptom is known as the Filatov or Belsky-Filatov sign. The same symptom was described somewhat later by an American doctor, Koplik. Notwithstanding the priority of the Russian doctors, the symptom is known in foreign (and in pre-revolutionary literature) as Koplik's spots (or sign).

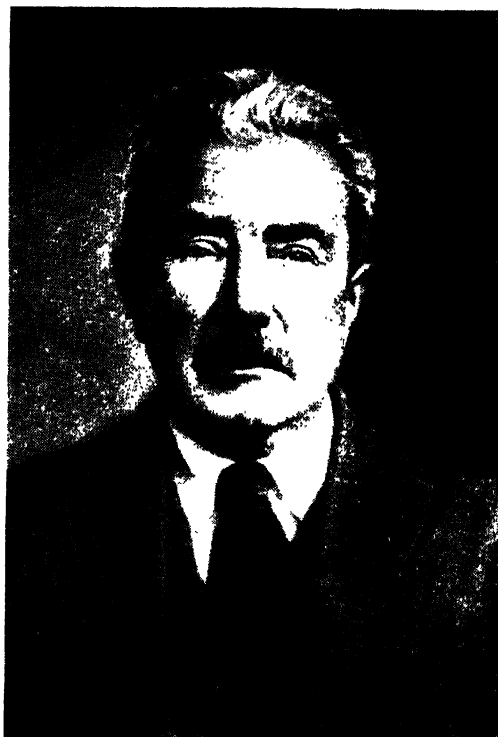
Filatov was the first to describe a hitherto unknown children's disease which he called "scarlatinous rubella", described fifteen years later by the British doctor Dukes, who called it the "fourth disease". Filatov also described a then little known disease of the lymphatic system, which he called "idiopathic inflammation of the cervical lymph glands". Three years later the same disease was described in Germany by Pfeiffer as "glandular fever". At present Soviet hematologists call it infectious mononucleosis, or "Filatov's disease".

Filatov is justly regarded as the father of Russian pediatrics.

N. Gundobin (1860-1908), professor at the Military Academy of



N. Gundobin



A. Kisel

Medicine in St. Petersburg, was the first pediatrician to go beyond the clinical observation of sick children and, with his pupils, to make a detailed study of the specific anatomical and physiological features of childhood. He set forth the results of his and his pupils' work in his comprehensive *Specific Features of Childhood* (1906), a book which came to be used as a reference not only by all Russian pediatricians but also by doctors in other countries.

One of the most ardent proponents of a prophylactic direction in Soviet public health services, specifically in child welfare, was A. Kisel (1859-1938), professor at the Second Moscow Medical Institute. Even before the Revolution Kisel had pointed out in his lectures, papers and speeches the importance of natural factors (air, sun and water) in the prevention and treatment of children's diseases. He paid particular attention to the development of prophylaxis and treatment of tuberculosis, rheumatism and malaria among children. He founded the first rheumatism clinic for children

—the first not only in the Soviet Union but in Europe and headed this clinic to the end of his life. Doctor Kisel gave a detailed description of a type of TB in children which he called “chronic tubercular intoxication”.

Among Professor Kisel's most outstanding pupils were S. Fedynsky and A. Koltypin.

S. Fedynsky (1876-1926) specialised in diseases of early childhood. As a doctor on the staff of a Moscow hospital he began to study the biochemical characteristics of very young children and drew his closest associates into that work.

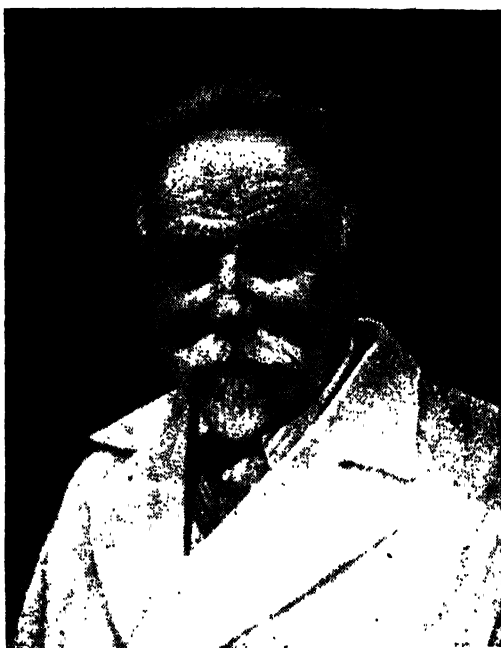
A. Koltypin (1883-1942) specialised in infectious children's diseases. He developed the teachings of Kisel and trained a number of scientific researchers and medical practitioners. He was the author of a textbook on children's infectious diseases.

N. Langovoi (1878-1947), another eminent pediatrician, wrote several books on diseases of early childhood. He was a champion of breast-feeding and of organisation of correct nutrition for infants.

V. Molchanov (1868-1959) was one of Filatov's closest pupils.



V. Molchanov



G. Speransky

His most important works deal with acute infectious diseases among children. (They include writings on the role of the adrenals in the genesis of toxic diphtheria, on the condition of the vegetative nervous system during scarlet fever and other acute children's infectious diseases, on the connection between scarlet fever and rheumatism.) In conjunction with his pupils Molchanov wrote a textbook entitled *Propaedeutics to Children's Diseases*.

Of contemporary Soviet pediatricians the most outstanding are Professors G. Speransky and M. Maslov.

G. Speransky (born in 1873), also one of Filatov's closest pupils, is well-known for his contribution to mother and child welfare in the Soviet Union. He has published many writings on nutritive and digestive disturbances in early childhood, on sepsis and pneumonia in infants. For his outstanding contribution to the development of medicine, particularly in the field of child welfare, Speransky was honoured with the title of Hero of Socialist Labour in 1957.

M. Maslov (born in 1885), professor at the Military Academy of Medicine and the Institute of Pediatrics in Leningrad, is success-



M. Maslov

fully developing Gundobin's studies of the age-group characteristics of children. He and his pupils have done valuable research on the biochemical features of healthy and sick children, and in a number of other important fields of pediatrics (diseases of the liver and kidneys, pneumonia, sepsis). His textbook on children's diseases for doctors and undergraduates has had several editions.

ANATOMIC AND PHYSIOLOGIC FEATURES OF CHILDHOOD

A child differs from an adult not only in the dimensions of his body, but also in the structure and functions of separate organs and systems.

The principal feature of childhood is a continuous growth and development, the constant perfection of the organs. All this is connected with an analogous continuous development of the central nervous system, first of all the development of the cerebral cortex. At the moment of birth the central nervous system is not fully developed. A particularly characteristic feature is the functional irregularity of the cortex of the cerebral hemispheres and the

younger the child, the less functionally developed is his brain; in the process of growth and development the functional organisation of the cerebral cortex soon matures.

At the same time the receptor systems of the child are also functionally immature and they gradually and consecutively attain functional physiological maturity.

Notwithstanding the fact that the brain of the newborn is relatively very large (as compared with body weight it is five times larger than the adult brain), yet its functional and morphological organisation are incomplete at the time of birth, in distinction from adults, in whom the brain acquires its complex structure and functions as a result of the protracted evolutionary process of development.

The spinal cord is more perfect; at birth the child already possesses the faculty of accomplishing primary reflective movements. But the nerve tissue of the brain is still immature, particularly the cortex which is the most important part of the brain, the centre of the higher nervous activity. That the newborn infant does manifest itself as a living creature in its movements and in certain in-born reflexes is due to the functions of the cerebral centres lying in the subcortical layers of the brain.

The brilliant Russian scientists I. Sechenov and I. Pavlov in their teachings on the physiology of the nervous system held that the entire complex of human activity is displayed in the development of a number of inborn (unconditioned) and acquired (conditioned) reflexes.

The child is already born with such food reflexes as sucking and secretion of saliva; these reflexes are called inborn or unconditioned, as are the motor reflexes he possesses while still in the womb. The cerebral cortex does not participate in these reflexes. They depend only on the receptors on the surface of the mucous membranes or the skin; these receptors convey the stimulus directly to the cerebral motor centres from which an order, as it were, is transmitted along the fibres of the motor nerves for a certain movement, or for the secretion of digestive juices by the corresponding organs. The cerebral cortex has nothing to do with these actions issued by the central nervous system. In the process of growth and development the child comes into closer and more frequent contact with his environment, and he begins to receive various gustatory, sound, light, and other stimuli. Gradually, as the cerebral cortex becomes more or less mature, its functions manifest themselves.

The child acquires new reflexes; these reflexes are called acquired, conditioned, or behaviour reflexes. While unconditioned reflexes are inborn and passed on from generation to generation, the acquired conditioned reflexes are developed during life and are strictly individual, although they may subsequently become fixed and inherited.

When the child is given food the latter irritates the nerve endings in the mucous membranes of the mouth and tongue, thus evoking the secretion of saliva and of other alimentary juices required for digesting food. Here the secretion of saliva is an inherited unconditioned reflex in which the cerebral cortex takes no part. But some time later the same food reflex (flow of saliva) appears even before the food has been tasted; it is called forth by the sight or smell of food the child has become accustomed to. In these cases the secretion of saliva is already an acquired reflex; its display does not call for direct stimulation of the digestive centre. First the vision or gustatory analyser-cells (receptors) in the brain cortex receive the stimulus which is then passed on via the conditioned arcs established in the brain between the cerebral cortex and the digestive centres in the medulla oblongata; from the latter centres the stimulus runs along the centrifugal nerve fibres to the salivary glands, and the flow of saliva commences.

Such conditioned digestive reflexes can be formed only on the basis of unconditioned reflexes; their formation occurs only if the child has previously seen or smelled the food several times directly before it was put into his mouth. If this coincidence of taking the food and perceiving its colour or odour has not occurred, that is, if the child has never seen or tasted this particular food, no conditioned food reflex—secretary reaction—takes place before the food is put into the child's mouth. Thus, if a child who has never tasted a lemon is shown one his attention will be attracted by the colour and shape, but no saliva will be secreted. But if the child has had several tastes of lemon excessive salivation occurs at the mere sight of the fruit. In adults, as we know, the simple mention or thought of a lemon can cause salivation.

In the subsequent life of the child, and later of the adult, numerous conditioned reflexes are established to the most diverse stimulations, coming both from the external environment and from the various internal organs.

Conditioned reflexes are temporary, sometimes they are easily elicited and disappear with the same ease. However, as was proved by Pavlov, certain circumstances in the process of the development of conditioned reflexes may convert them into inherited un-

conditioned reflexes. Studies performed by Soviet researchers have shown that the formation of conditioned reflexes may be observed toward the end of the prenatal (embryonic or fetal) period and after birth, particularly after the second month, but their range of formation is still quite limited.

From this time on the child begins to distinguish taste (sweet, salted, sour, bitter), reacts to some odours, begins to distinguish colours, and then faces (recognises its mother). The appearance of a response to taste and colour proves that the cerebral cortex has already become mature enough for the higher nervous activity of the child and for its further progressive development.

The teachings of the outstanding physiologist Pavlov on the higher nervous activity and on the role environment plays in the development of the central nervous system, and particularly of the cerebral cortex, have given us a greater insight into the dependence of the normal growth and development of the child, on the proper management of the external factors surrounding him, on the conditions in which he is brought up, on his care and proper nutrition.

The investigations of N. Krasnogorsky, one of Pavlov's pupils, have shown that the nutrition of the child largely depends on his reflexes, and therefore on the proper functions of the cerebral cortex. It has likewise been proved that children who do not receive normal stimuli from their environment, who live without the external impressions they require, are not only very backward mentally, but their physical development is also lower than normal.

The central nervous system, and first of all the cerebral cortex, the principal representative and distributor of all the functions of the growing organism, reacts very keenly to unfavourable conditions in the life and upbringing of the child; concern for the creation of normal conditions for the growth and development of children is one of the paramount objectives of our state and of Soviet society in general.

PERIODS OF CHILDHOOD

In order to study the physiological and morphological aspects of a normally developing child it has been found convenient to divide childhood into definite developmental periods—*stages of childhood*. This is also necessary for properly managing regimens and organising conditions for the normal upbringing of the child in accordance with age specificities.

The following age periods are distinguished: the neonatal or newborn period, infancy, early childhood, preschool age, early school age, later school age (adolescence).

The newborn stage starts the moment the umbilical cord is cut (the first 15-20 days of life). The infant begins adapting itself to a new environment; from this moment it ceases receiving oxygen, warmth and nutrition from the body of its mother. Its nutrition is now mother's milk; it receives oxygen through independent respiration; it keeps up its body temperature to a certain extent, although the thermoregulating system is still immature, and the infant begins to produce antibodies against various infections independently, not receiving them passively, as in fetal life. During the newborn period the umbilical cord withers and falls away.

Owing to the immaturity of the central nervous system, particularly of the cerebral cortex, all the functions of the child's body are still imperfect; at this stage of life the child requires careful attention, and his only food is breast milk.

The duration of the newborn period, that of adaptation to new conditions of existence, differs in individual children. There is no strictly defined boundary dividing this period from the following period of childhood; in some children adaptation takes somewhat longer than 20 days.

During *infancy* (up to one year of age) growth is very rapid, as is also the development of all the bodily systems and organs. Therefore, this period of development calls for a much greater amount of food as compared to body weight; but at the same time the functions of the digestive organs are still immature, so that the slightest violations of feeding routine at this age easily lead to all kinds of digestive and nutritional disorders.

In infancy the central nervous system, particularly the cerebral cortex, becomes more and more mature as compared with the newborn phase.

During this period the child still requires mother's milk for his proper development. In infancy the child begins cutting his teeth and he gradually becomes adapted to food other than breast milk (cereals, fruit juices, vegetables).

In *early childhood* (from one to three years) the child no longer requires breast milk. During this stage of development the static functions are finally developed and the milk teeth are all cut; with the rapid development of the cerebral cortex the system of speech begins functioning, and the child utters whole phrases, not just separate words, mastering the art of speech.

During the period of *preschool age* (from three to seven years) the development of the child's organs and systems continues, his mental powers progress, the further development of cerebral functions becomes stabilised.

During *early school age* (from seven to twelve years) the primary or milk teeth are replaced by secondary or permanent teeth, and the majority of organs attain complete development.

Later school age, or adolescence (from twelve to eighteen years) is the time of sexual maturation. During this stage the functional organisation of the brain attains its complete and manifold perfection. The thymus undergoes atrophy, while the sexual glands, or gonads, begin functioning intensively. The nasopharynx becomes larger, the voice changes.

The proper development of the child in all periods is connected closely with hygiene, domestic conditions, and upbringing.

Weight, Height, Circumference of Head and Chest

The weight of a woman's first child is usually lower than that of her subsequent children. An important part is played in this matter by social conditions. Thus, it has been observed that women who have maternity leave before childbirth usually give birth to infants that are larger and more developed.

In the first 3-5 days of life the child loses approximately 7-9 per cent of his initial weight (150-300 g), then he starts gaining weight, and attains birth weight on the 10th-12th day.

The loss in weight that is regularly observed in almost all infants is called physiological loss. The greater part of this loss (about 75 per cent) may be explained by the fact that after the exceptionally favourable conditions experienced in the womb the baby finds itself in absolutely new external surroundings, to which it cannot immediately become adapted. In this new environment the infant's skin and lungs emit a considerable amount of water. Some share of the physiological loss in weight should be attributed to the excretion of primordial feces (meconium) and urine (10 to 20 per cent), and also the possible vomiting of swallowed amniotic fluid. One must also take into consideration the fact that the mother may not produce enough milk the first days after delivery, as lactation is only developing at this time.

In many instances this "physiological" loss of weight may be significantly decreased if the newborn is given enough water and food in the first 24 hours of his life. The loss in weight will be considerably less if the child is put to the breast 12 hours after

birth and not 24, as was formerly customary. The children of women who have born several children lose less in weight, as lactation is established earlier.

A weight loss exceeding 300 g should be considered abnormal, signifying that the child is not getting enough food, or that he is ill.

After the initial weight has been recovered by the 10-12th day of life, a healthy infant begins gaining weight rapidly; this increase is particularly high in the first months of life—25 to 30 g a day. Then it gradually slows down and in the last months of the first year of life the child gains 10 to 15 g per day. The average monthly weight increase in the first six months of life is 600 g, in the next—500 g. Proceeding from this, the weight of an infant may be determined by the following equation: weight at any given month of life is equal to birth weight plus 600 g (or 500 g) for each month of life.

For instance, a three-month-old baby will weigh: $3,500 + (600 \times 3) = 5,300$ g.

By five-six months the baby doubles its birth weight, by one year the weight has trebled, and the child should weigh something like 10,000 g at this time.

Normal weight increase is affected by the child's surroundings, the method of feeding, and also individual traits.

Mixed or bottle feeding, insufficient fresh air and light, unfavourable housing and sanitary conditions, various diseases—all this may affect correct weight increases and the general development of the child.

It has also been noted that weight increase is irregular in the various seasons of the year: at the end of summer and in the autumn children gain somewhat more than they do in the winter and spring.

Under normal conditions of feeding and care babies born with lower weights, or prematurely, gain more rapidly than larger children do: they double their birth weight by the third or fourth month of life, and treble it by six months.

The weight of children over a year increases much more slowly than in infancy. During the second year of life the child gains 2.5 to 3 kg (200-250 g a month); from three to six years the yearly weight increase is 1.5 to 2 kg. As compared with the age of one year the child's weight doubles by the time he is six or seven, becoming 20 kg, and doubles again by thirteen or fourteen years.

In order to determine the average weight of a child over a year old his weight at one year is added to the number of years he has lived multiplied by two (the average yearly weight increase is 2 kg).

Thus the weight of a six-year old should be $10 \text{ kg} + (6 \times 2)$, that is, 22 kg.

The rate of the weight increase of children over a year old is not uniform in the different periods of childhood. It becomes higher as puberty approaches.

The yearly weight increase in boys of 14-15 attains 3 to 5 kg, while by 16-17 it is 5.5 to 8 kg.

The rise in the yearly weight increase is manifested earlier in girls, between 9 and 12 (2.5-4.5 kg), becoming 5 to 8 kg by the time the girl is 13-15; however, after 16 the yearly increase falls and girls subsequently weigh less than boys (Tables 1 and 2).

The *height*, that is, the length of the child's body from heel to crown increases by half in the first year of life: if the length of a newborn infant delivered at term is approximately 50 cm, then by the end of the year he will grow about 20-25 cm. The monthly increase in the length of infants is:

in the first quarter of the year	3 cm
in the second quarter of the year	2.5 cm
in the third quarter of the year	1.5 cm
in the fourth quarter of the year	1 cm

(see Table 3)

By the end of the second year the increase in height is 10-12 cm, in the third year it is 7 cm, and later the rate of height increase becomes still less. The average annual height increase is 5 to 6 cm.

The height of a child over a year old is defined by multiplying the number of his years by 5 or 6 cm and adding the product to 75 cm (the height of a one-year old).

Thus the height of an eight-year old should be: $(5 \times 8) + 75 = 125$ cm. At five the height of the child is double of what it was at birth, and triple at 14-15 years of age.

The following factors are necessary for the attainment of proper stature: 1) normal sanitary conditions; 2) breast-feeding in the first year of life; 3) full-value food in subsequent (after infancy) periods. Children living in poor sanitary conditions are often smaller than other children. Children nursed on cow's milk are likewise smaller than breast-fed infants. A good stature requires a sufficient amount of proteins and vitamins. Height is inhibited by various infectious diseases, both acute and chronic. Nutritional disorders and rickets also inhibit height increase. Three periods of accelerated gain in height are defined in childhood: 1) in the first year of life; 2) from 6 to 8; 3) during puberty; in girls from 12 to 14, in boys from 14 to 16. The seasons of the year are also

Table 1

**Weight of Child in First Year of Life (in grams)
(according to Orlov; figures are approximated)**

Age (months)	Boys	Girls
Newborn	3,100-3,400	3,000-3,200
1	4,000	3,750
2	4,850	4,600
3	5,600	5,250
4	6,350	6,000
5	7,050	6,600
6	7,650	7,200
7	8,100	7,550
8	8,600	8,000
9	8,900	8,200
10	9,260	8,600
11	9,400	8,900
12	9,800	9,100

Table 2

**Average Weight of Children from 1
to 17 Years of Age (in kilograms)**

Age	Boys	Girls
1	9.8	9.5
2	12.0	11.6
3	14.1	14.2
4	15.9	15.3
5	17.9	17.8
6	18.8	19.8
7	22.1	21.4
8	23.9	23.2
9	25.6	24.8
10	28.1	27.5
11	30.5	30.0
12	33.4	33.8
13	36.4	38.5
14	41.7	43.7
15	46.8	47.3
16	52.9	51.2
17	58.0	53.2

Note: The weights are cited according to Salistovskaya (for children under three), Korsunskaya (for children between three and seven), and Aron (for children over seven).

important. From the middle of August to the middle of December there is a sharp increase in weight, while gain in height is slow; from the middle of December to the middle of April gain in both weight and height is moderate, and from the middle of April to the middle of August height increase is rapid while weight increase is negligible. The intensive summer gain in height is probably due to the child being outdoors more, in the sun and air. The low weight gain is probably due to the fact that the child is much more active than in the winter, drinking more and eating less.

There are special scales for weighing young babies.

The length (height) of infants is measured on a horizontal measuring board (Fig. 1). Older children are measured on a vertical measuring board (Fig. 2).

The *skull* of a baby delivered at term usually has only one open spot—the anterior fontanel—between the frontal and parietal bones of the cranium. It has the shape of a rhombus 2-2.5 cm in diameter, and closes by the time the child is 12-15 months old.*

To gain a correct conception of the physical development of the child it is necessary to know, besides his weight and height, the circumferences of his head and chest, the width of his shoulders, length of extremities.

Shoulder width at all ages is one quarter of the height.

The circumference of the head is 33-35 cm at birth, 1 to 2 cm greater than that of the chest, which is 31-34 cm in

Table 3

Crown-Heel Length (Height in cm) of a Baby in Its First Year of Life (after Orlov)

Sex	Length of newborn	Length at end of month of life											
		1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th	12th
Boys	49.52	53.9	57.1	60.1	62.1	64.4	66.3	67.7	69.4	70.7	71.7	72.6	74.4
Girls	48.51	52.9	56.8	58.8	61.0	63.1	65.0	66.2	68.0	68.8	70.2	71.1	72.8

* The fontanel is measured along its medial line, from the middle of one side to the middle of the other, not from corner to corner.

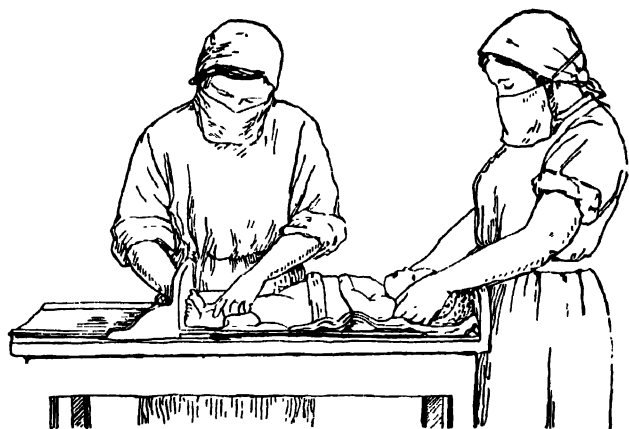


Fig. 1. Measuring infant's height (crown-heel length)



Fig. 2. Measuring height of child older than one year

circumference. During the first year both head and chest grow intensively. By six months the circumferences of head and chest become equal, 43 cm, after this the chest circumference becomes larger than that of the head; at the end of the first year the chest measures 47 cm, the head 46. After one year the head grows very slowly, while the chest circumference increase becomes more and more noticeable as compared with the head. Thus, at five the head circumference is 50 cm, the chest 56 cm; at ten years the figures are respectively 52 and 63 cm, at 16—54 and 63, and at 18—54 and 79 (Figs. 3 and 4).

The arms and legs of the newborn are almost as long as the trunk (18-19 cm). According to Professor Maslov's data the legs grow threefold by seven years, the arms two and a half times, and the trunk two times; by 16 the legs have become five times longer

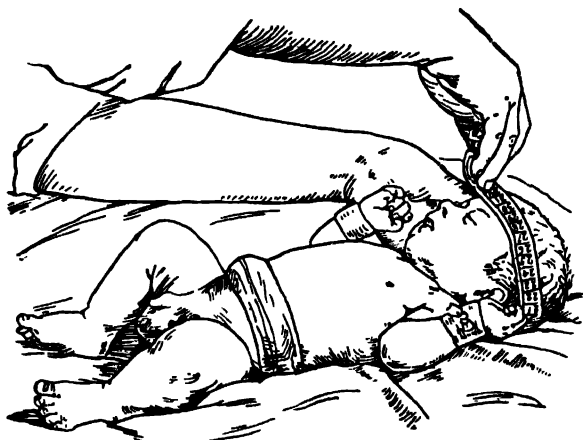


Fig. 3. Measuring head circumference

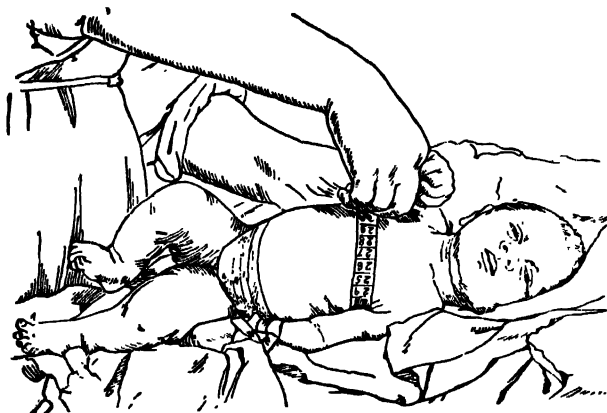


Fig. 4. Measuring chest circumference

than they were at birth, the arms four times, and the trunk three times. In prenatal life the legs of the fetus are bent to its stomach, hence they become curved laterally owing to the softness of the bones; this is so-called physiological bow-leggedness. The small bones of the newborn's hands and feet are cartilaginous, and only gradually do points of ossification appear that are well defined on X-ray pictures.

Primary and Secondary Dentition

The eruption of the *milk teeth* (primary dentition) begins at approximately six months.

Usually the first two teeth to be cut are the lower central incisors, in the seventh month; the upper central incisors come a month later, so that by eight months the baby has four teeth. In the following four months the other four incisors are cut, and the remaining 12 milk teeth erupt during the second year (Fig. 5).

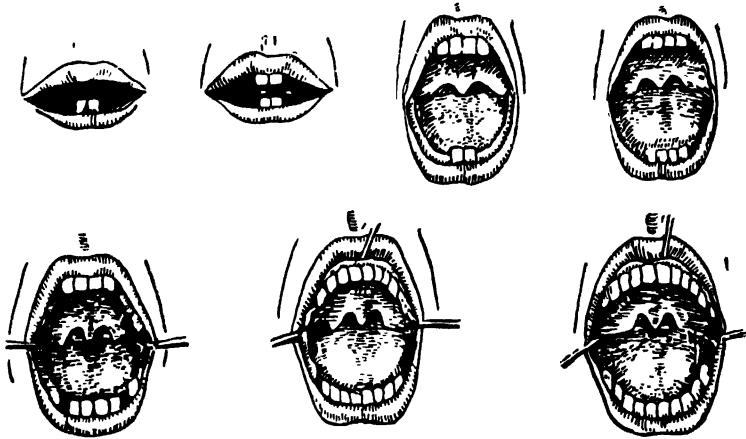


Fig. 5. Eruption of milk teeth

Delayed dentition points, in most cases, to some disorder, most frequently rickets, myxedema, or the Down disease (mongolism).

The eruption of the permanent teeth takes place in the 5th to 7th year of life; the first to appear are the third upper and lower molars. Later, between six and twelve years, the primary milk teeth fall out in the order of their appearance, and they are replaced by permanent teeth. Between 12 and 14 the fourth pair of molars erupts while the fifth pair (the so-called wisdom teeth) appears by 18 to 20 years.

The teeth begin growing at birth; the soft tissues covering and surrounding them are gradually resolved, so that by the end of this process the tooth comes through easily and painlessly.

Formerly many diseases were blamed on teething. Now it is known that no diseases are connected with dentition; this is a harmful superstition that should be combated, as it interferes with the timely diagnosis of a true disease.

Specific Features of the Skin, Subcutaneous Tissues and Muscles

The skin. The skin of the newborn is bright red and is covered by a primary greasy substance. After a few days the redness (physiological erythema) disappears and during the following several days the epidermis is shed in flakes or layers (desquamation). In general infant skin is soft and tender, with a thin epithelium and weakly developed cutaneous layer; the vascular system is highly developed: there is a strong tendency to desquamation and the formation of crusts (most of all on the scalp).

The sebaceous glands start functioning in the prenatal period, so that an excessive secretion of fat may be noted on the surface of the skin; often accumulations of fat may be seen under the epidermis on the wings of the nose, occurring as yellowish-white spots.

The sweat glands are, on the contrary, immature at the time of birth, and the child does not perspire in the first weeks of life. The glands gradually become active, and by 4-5 months they function as in adults. This weak function of the sweat glands is connected with the immaturity of the central nervous system, particularly of its sweat-secretion centres.

The hair of the newborn infant is fairly well developed, sometimes even being quite fluffy, although it often falls out soon; having no shaft, the hair of the newborn is very soft.

The subdermal layer of fat is sufficiently developed in the newborn, noticeably expanding in the first six months.

This layer grows first of all on the face and extremities, then on the trunk and abdomen. When the child loses weight the fat disappears in the reverse order.

In the period between three and eight years the fat layer does not increase, but subsequently, particularly in girls approaching puberty, it accumulates again intensively.

The body fats in early childhood are characterised by a high palmitic and stearic acid content, having higher melting points than other fats. For this reason in the first days and weeks of life the subdermal layer of fat in children sometimes hardens quite easily under the effect of cold, or owing to some severe disease (sepsis, toxicoxis).

The nervous apparatus of the skin (skin receptors), as well as the central nervous system of babies in general, is still insufficiently developed and the skin functions are in many aspects immature. As the central nervous system gradually and consecutively devel-

ops and becomes differentiated, particularly the cerebral cortex, the functions of the skin also develop and improve.

The function of the skin is very complex. It lies principally in the *protection* of the organism against harmful external influences.

This protective function of the skin is not displayed in early childhood; the upper layers of the skin are very delicate and therefore easily injured, abrasions and scratches may be an inlet for any kind of infection. All manner of toxic substances are easily absorbed through the thin derma, in the lower layers of which runs a network of blood vessels; microbes (particularly pyogenic) also penetrate it easily, and this can cause general sepsis. This is why one must be careful about using various irritating salves and ointments (turpentine, iodine).

However, the skin of children, as compared with adults, possesses a high restorative quality; granulation and epithelisation of wound surfaces is much faster in children than in adults.

The respiratory function of the skin is expressed mainly in the emission of carbon dioxide and water into the surrounding atmosphere. It is known that the elimination of the respiratory function of two-thirds of the skin by application of some substance impermeable to water or air may cause death, as in suffocation.

The respiratory function of the skin plays a much greater part in the life of children than it does in adults.

The skin is also an important organ of *heat exchange* (thermoregulation); however, owing to the immature thermoregulating functions of the central nervous system in children, and the intensive emission of moisture and heat through the surface of the skin, this function is extremely insufficient. As we know, in adults the so-called pilomotor reflex—erection of the hairs of the skin (goose-flesh)—is displayed in response to excessive chilling as a signal of the cerebral cortex: the skin contracts with the contraction of the hair muscles, taking on the appearance of plucked goose-flesh; in this manner the total surface of the skin decreases, and so heat emission decreases too. Excessive warmth is regulated by another mechanism: the function of the sweat glands is intensified, evoking increased secretion of sweat on the surface of the skin, and its evaporation cools the skin.

The pilomotor reflex is absent in the first days of life, newborn infants have no "goose-flesh", and, moreover, the functions of the sweat glands are absent or very weakly expressed; therefore, infants rapidly become cold if the air is cold, or, on the other hand, they are easily overwarmed as their bodies cannot emit the excessive warmth by intensive sweating and evaporation of the sweat.

There is another important function of the skin—the formation in it of vitamin D₂, a factor facilitating proper ossification of the skeleton.

The *muscles* of newborn infants are developed poorly. They comprise about one-fourth (25 per cent) of their body weight, while in adults they take up 40-43 per cent of this weight. The muscle fibres of children are significantly thinner than in adults. The increase in the muscular mass during the child's growth is due to the growth of the muscular fibres, and not to an increase in their number. The motor faculty of the muscles is manifested in children at first in the muscles of the neck and body, and later in the extremities.

Muscular strength and tonicity are low in children. In the first months of life a higher tonicity is observed, so-called "physiological hypertension" due to a specific function of the central nervous system. In older children increased muscular tension is a symptom of some pathological condition in the central nervous system.

Low muscular tension (hypotension) in babies is due to rickets.

The development of the child's static functions depends on the development of his central nervous system. Proper care and training is an important factor in the stimulation of the baby's movements.

The Skeleton and Bones

The bones (the osseous tissues) of the newborn contain more water and less solid substances than do the bones of adults. In the fetal period skeletal ossification is a rather late process, and at birth the infantile skeleton still contains a great deal of cartilaginous tissue (the spinal column, the wrist). The structure of the harder part of the skeleton, consisting of already ossified tissue, still differs from the bones of adults; in children this structure is fibrous, in adults it is laminated; there are less salts in children's bones, therefore they are more flexible and resilient than in adults, and not so hard.

Babies' bones are easily deformed under undue pressure (tight clothing, narrow footwear, improper carrying in arms). The bones of children afflicted by rickets are deficient in calcium and phosphorus salts, so that in some parts of the skeleton a softening of the bones may occur. This is conducive to deformation (softening of the bones of the back of the head, the chest, the limbs).

The *head* of the newborn and of infants is large, comprising one-fourth of the length of its body (in adults it is only one-seventh to

one-eighth). The edges of the skull bones do not converge closely; a divergence of the bones may be felt upon probing with the fingers, particularly in premature infants.

By the second or third month the edges of the skull bones join up, and suture tightly by the age of three or four. At the point of juncture of the frontal and two parietal bones the anterior fontanel may be felt; it is rhomboid in shape, its lateral and posterior ends turning into loosely knitted seams. In the back of the head, between the two parietal bones and the occipital bone lies a posterior fontanel; however, it is not always discernible (only in 25 per cent of infants). This fontanel is triangular in shape.

The anterior fontanel is measured from the middle of one side to the middle of its opposite (through the medial, not the horizontal, line of the rhombus). The usual dimension of the anterior fontanel is 2.0×2.5 cm, but it may be larger or smaller.

The posterior fontanel, if it is present, closes very soon, by the second month of life, while the anterior fontanel only closes by 12-15 months. The closure of the fontanel is delayed in rickets and hydrocephaly (dropsy of the brain). In premature infants two more fontanel, besides the ones already mentioned, may be found on the sides of the skull.

The child's head is round, but in rickets the frontal and parietal tubercles grow excessively and the head takes on a square shape (*caput quadratum*), the head itself also becoming larger. The head is much larger in children afflicted with hydrocephaly. This disease develops as a side effect of meningitis: an excessive accumulation of fluid occurs in the cerebral ventricles; pressing on the cranial bones, the liquid separates them (hydrocephaly, water on the brain). The opposite may occur if the brain develops insufficiently in the fetal state. Then the skull, chiefly the cranium, stops growing from the very first days after birth, the fontanel close too soon, and the head is small as compared to normal (*microcephaly*).

The *spinal* (or *vertebral*) *column* of the newborn is straight and consists of cartilaginous tissue; only subsequently does a gradual ossification of the vertebrae occur.

From the moment the child begins holding its head the first physiological deformation appears—curvature of the neck or cervical lordosis. When the child sits up, at six or seven months, a second curvature is formed in the thoracic region—kyphosis of the thorax, and when he begins to walk a curve is formed in the lumbar region—lumbar lordosis.

In later life, under the influence of unfavourable conditions (bad posture at the table at home or the desk at school) the spine may

become deformed laterally—scoliosis—or, under the effect of tuberculous lesions a sharply defined hump may grow—kyphosis.

The thorax (chest) of the newborn looks like a truncated cone, the ribs are uplifted at right angles to the spinal column, so that the chest is in the inhaling position. This limits movement, does not allow the lungs to expand much; therefore, the breathing of children is rapid and shallow. Gradually, as the child begins to walk, the configuration of his chest changes and its respiration capacity increases. The final formation of the chest occurs at 12-13 years, when it takes the shape it will have in adult life (Fig. 6).

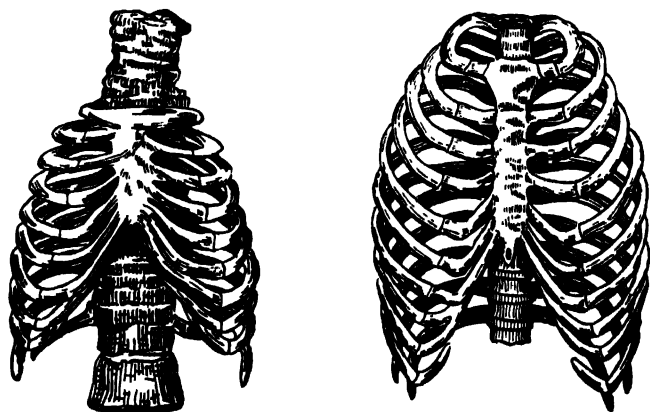


Fig. 6. Chests of children and adults

In children *the tubular bones of the extremities* grow by gradual ossification of their cartilaginous end—the epiphysis. Points of ossification first appear in the depth of the epiphysis, and subsequently join up with the shaft of the tubular bone, the diaphysis. The growth of the long (tubular) bones continues for a prolonged period, ceasing only at the age of 25.

The pelvic girdle is the same in infants of both sexes, and only at the age of 6-7, or even later does the pelvic girdle in girls become wider than in boys.

The Respiratory Organs

In prenatal life the lungs of the fetus are in a collapsed state—atelectasis.

Although it has been proved that intrauterine respiration exists, and that every respiratory movement evokes negative pressure in

the thoracic cavity, this pressure is so insignificant that it cannot expand the lungs completely. Only after the umbilical cord has been cut and tied, and the infant's blood has become very deficient in oxygen, does the negative pressure in the lungs become so great that it causes the lungs to expand completely (the first breath).

The expansion of the lungs by the first breath the child takes stimulates the vagus receptors in them, and thus rhythmic pulsations and cerebral regulation of respiration are established.

The nose of the newborn child is small, flat, its bridge fallen. The nostrils are narrow, and the delicate nasal mucosa is very rich in blood vessels. The pharynx and larynx are also very narrow, the nasal cavities are still in a rudimentary state, and the development of the frontal cavities commences only in the second year of life. As the nasal ducts are very narrow, even a slight cold may impede respiration, causing short breath and dilation of the nostrils. The nasal cavities develop and approach adult structure very gradually. The pharyngeal cartilage is very soft; the mucosa of the pharynx is very tender, its vascular network well developed, so that even a slight swelling of the mucosa impedes respiration.

The thorax of the newborn is barrel-shaped and short: the ribs are at right angles to the spinal column; owing to this the respiration of the newborn is of the abdominal or diaphragmatic type. When the child begins to sit and walk the lower part of his chest descends, the ribs take an oblique angle to the spinal column and become very curved; thus the capacity of the chest increases. As the lungs of the newborn are comparatively large, filling the entire thoracic cavity, and the ribs are soft, deep breathing is impossible; the required amount of oxygen is supplied to the tissues by means of accelerated breathing (40-60 respirations per minute). A six-month-old child takes 35 to 40 breaths a minute. As the dimensions of the thorax increase the child is able to take deeper breaths: by 12 months the number of respirations per minute goes down to 30-35, becoming 25 by the time the child is 5-6 years old, his breathing is thereafter of a mixed type, both costal and diaphragmatic. At the age of 12 to 15 the number of respirations goes down to 18-20 per minute; in boys the abdominal type of respiration prevails, in girls the costal. The bronchi of children are narrower than in adults, and therefore the slightest swelling of their mucosa greatly impedes breathing.

The expiratory breath sounds in infants differ from those of adults or of older children. Babies' breathing is harsher and at times a weak expiration is heard. This type of respiration is called puerile (juvenile) respiration, while in adults normal breathing is

softer, more low-pitched—vesicular respiration—and the expiration is not heard.

This type of breathing in children depends on the anatomic structure of the pulmonary tissue in which there are more connective tissue seams than in adults; it is to this that the difference in the pitch of sound is due when the lung as a whole vibrates. Besides this, the thorax walls of children are thinner than in adults, so that sounds are conveyed better. The lung sounds are also affected by the narrowness of the bronchial ducts in children, the shorter length of their windpipe (the trachea), and also by the small dimensions of the chest. All these factors create the specific aspect of normal breathing in early childhood.

The narrowness of the tracheal and bronchial ducts often causes severe respiratory difficulties at the slightest swelling of the bronchial mucosa in very young children.

Normal development of the respiratory organs in childhood (the chest and lungs) depends on proper management and care. When the child spends most of the time in stuffy premises, getting but little fresh air and sunshine, normal development is obstructed, and hence the respiratory organs do not develop properly. All this may lead to various disorders of the respiratory system (laryngitis, bronchitis, pneumonia).

In order to prevent such diseases parents and child-care personnel should strive to make the most of fresh air (frequently airing premises, outdoor walks, no washing, cooking, nor smoking in rooms where children are).

The Blood Circulatory System

Prenatal blood circulation differs widely from postnatal circulation; it is effected through the umbilical cord. The latter consists of two arteries and one vein lying in a matrix of embryonic tissue (Wharton's jelly), sheathed by the amnion. The oxygenated blood is delivered from the mother's circulatory system to the fetus through the umbilical vein, passing into the portal vein and the inferior vena cava (via the duct of Arantius, or the ductus venosus), then into the right atrium, from where it passes through an oval opening into the left atrium down to the left ventricle, and finally into the aorta from which it is distributed to every part of the body. The venal blood returns via the umbilical arteries to the placenta; only a very small part of the blood reaches the right ventricle and the pulmonary artery and from there through the ductus arteriosus (Botallo's duct) flows into the aorta. After birth

the umbilical vessels close up within the first three months of life, then the ducts of Arantius (ductus venosus) and of Botallo, and, finally, the oval opening connecting the atria (Figs 7 and 8).

The *heart* of the newborn is comparatively large and its walls are thin. Owing to the high position of the diaphragm the heart lies horizontally. Subsequently, as the diaphragm descends, the position of the heart changes. The ventricles of the heart are large, and the orifices of the larger vessels are wide. The heart grows most significantly in two periods of life—the first year and puberty. The walls of both ventricles are almost of the same thickness, but by the end of the first year of life the walls of the left ventricle become twice as thick as the walls of the right one. As the intercostal spaces are very narrow up to the age of two, the pulsation of the heart is probed with difficulty in the fourth intercostal space, 1 cm to the left of the midclavicular line. After two years the pulsation is easily felt on the midclavicular line. The heart sounds are louder and shorter in children than in adults. Emryocardia is normal in the newborn, i. e., a condition in which the first and second heart sounds (systolic and diastolic) are almost identical. The *pulse* is frequent, sometimes irregular. According to Filatov the pulse rates over 100 beats per minute in children younger than six years, after six it becomes less frequent; the number of pulse beats per minute in infants is approximately 140, in the second year of life 120, and by 16 years it falls to 80. The pulse beats four times for every respiration.

Arterial openings in children are comparatively wider than in adults, veins and arteries are of approximately similar widths, while in adults the veins are much wider than the arteries.

The greater width of the vascular system as a whole in children makes their *blood pressure* lower than that of adults. Blood pressure values are usually determined after the Molchanov equation: blood pressure at a given age is $80+2n$, where “*n*” is the child’s age in years.

The amount of blood per kg of body weight is much greater in children than in adults (in the latter it is 50 g, while in an infant it is 100-110 g); circulation time in children is only half of the time required for the blood to circulate in adults.

In children cardiac activity and blood circulation are more precise than in adults, as the heart itself and the vessels, as well as the nerves of the cardiovascular system have none of the pathological changes that may be present in adult life owing to the harmful effects of all manner of intoxication (alcohol, tobacco) and of various infectious diseases.

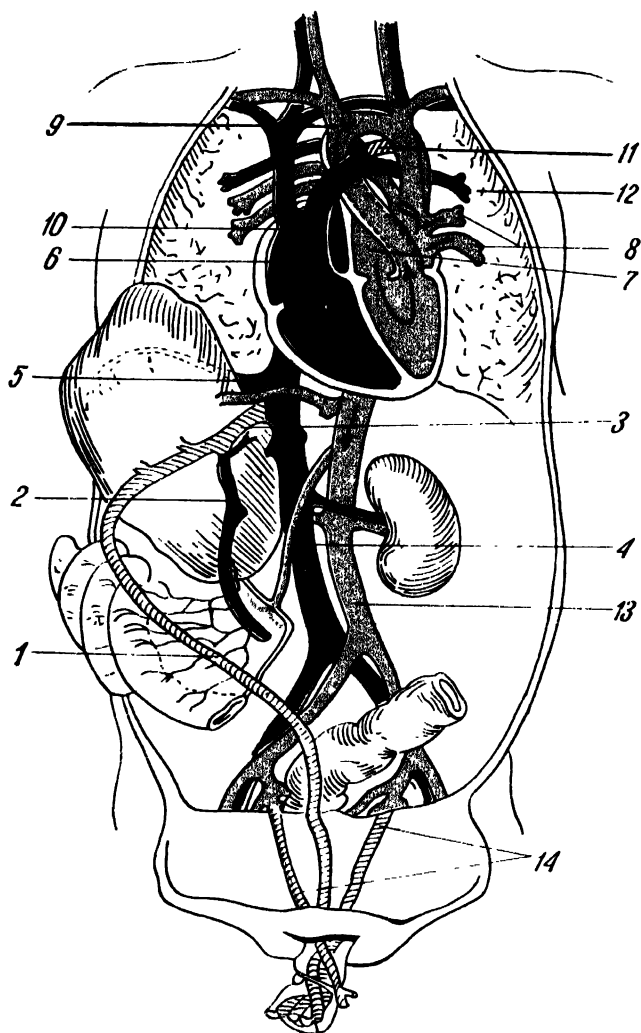


Fig. 8. Circulation in the Newborn

1—umbilical vein; 2—portal vein; 3—duct of Arantius; 4—inferior vena cava; 5—hepatic vein; 6—right atrium; 7—left atrium; 8—pulmonary vessels; 9—aorta; 10—pulmonary artery; 11—Botallo's duct; 12—lung; 13—descending aorta; 14—umbilical arteries

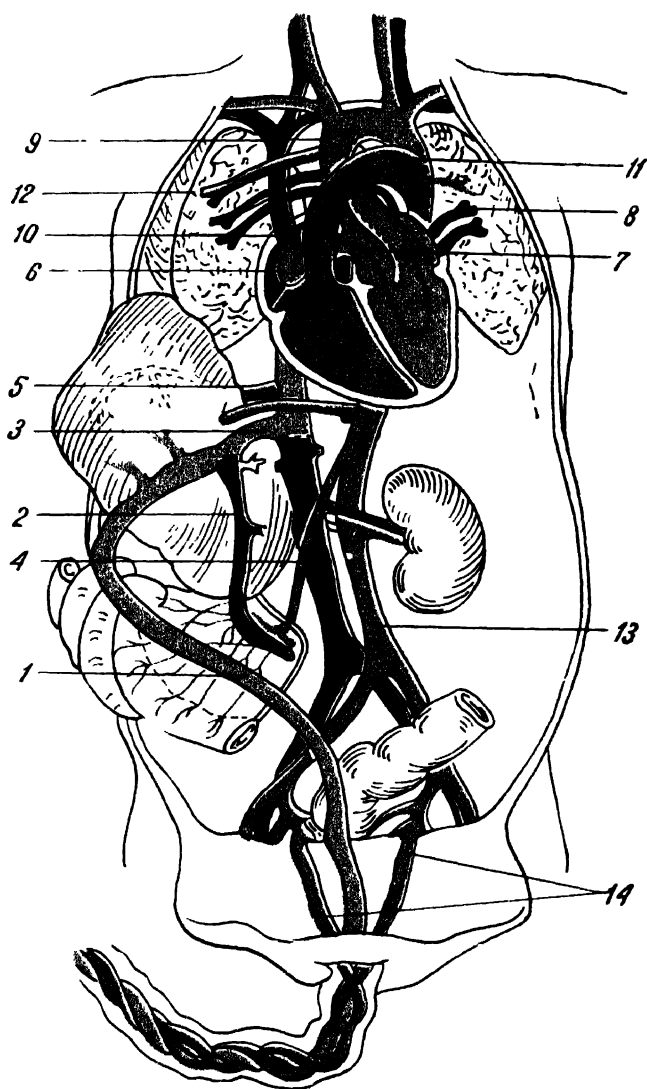


Fig. 7. Fetal Blood Circulation

1—umbilical vein; 2—portal vein; 3—duct of Arantius; 4—inferior vena cava; 5—hepatic vein; 6—right atrium; 7—left atrium; 8—pulmonary vessels; 9—aorta; 10—pulmonary artery; 11—Botallo's duct; 12—lung; 13—descending aorta; 14—umbilical arteries

Children's cardiovascular systems usually function properly in such diseases as pneumonia, and they do not often require the administration of various cardiac medicines, while pneumonia in adults frequently does call for such drugs.

Blood and Blood Formation

In fetal life the organs of blood formation, or hemopoiesis, are the liver, spleen, marrow and lymphatic tissue. The marrow starts functioning only during the second half of fetal life, to become finally developed by the time of birth; it is the principal site of formation of the blood cells; the spleen and lymphatic tissues are secondary in this respect.

Nucleated erythrocytes and physiological lymphocytosis are frequently observed in the systemic blood of normal newborn infants; this is probably due to the intensive activity of the lymphatic tissue.

High hemoglobin levels and erythrocyte and leukocyte counts in the newborn are due to a thickening of the blood caused by loss of water. The blood of infants is usually poorer in erythrocytes and hemoglobin than that of adults, and richer in leukocytes.

In very young children blood is formed in all the bones, but beginning from the age of four the red marrow of certain bones turns into yellow marrow in which the hematopoietic functions have been lost, and the cells are largely replaced by fat cells. By the age of 12-15 years hematoipoiesis occurs in the red marrow remaining only in the flat bones and in the epiphyses of the long (tubular) bones. In the first hours after delivery the blood of the newborn shows a very high hemoglobin level (110-140 per cent by the Sahli hemoglobinometer) and an increased erythrocyte count, (6,000,000 to 7,000,000); a decrease in hemoglobin and erythrocytes occurs on the very first day of life. By the end of the newborn stage the hemoglobin level goes down to 90-100 per cent and by the end of the first year becomes, on the average, 70-80 per cent; by 12-15 years the hemoglobin rises to 82-86 per cent. The erythrocyte count falls to 4,600,000 by the end of the first month of life.

The leukocyte count at birth is 10,000 to 30,000; in the first hours after birth there appears to be a certain tendency to an increase of this count, but by the tenth or twelfth day the number of leukocytes falls to 9,000-10,000. It is characteristic of the newborn that the differential leukocyte count in the first hours of life shows 65 per cent of neutrophils; however, by the fifth-sixth day of life the percentage of neutrophils and lymphocytes becomes

equal, and later the percentage of lymphocytes increases, attaining 61-62 by the end of the first year. Then the neutrophil count again increases, and becomes equal to the lymphocyte count by 5-6 years, after which the number of lymphocytes falls, owing to an increase in neutrophils; by 13-14 years the blood values are the same as in adults (Table 4).

Table 4

**Blood Values in Children at Various Age Levels
(as compared with the composition of adult blood)**

	Newborn	Infants	Older children	Adults
Hemoglobin (per cent) . .	100-140	75-85	80-90	100
Erythrocytes (millions) . .	5-8	4.5-5.0	4.5-5.0	5.0
Leukocytes (thousands) . .	12-20	8-12	7-10	6-8
Blood platelets (thousands)	100-400	200-300	200-300	200-300
Neutrophils (per cent) . .	60-70	15-40	gradual increase to 60	50-65
Lymphocytes (per cent) . .	20-30	55-75	gradual decrease to 35	20-35

The Lymphatic System

An extensive network of *superficial lymph nodes*, or glands, exists in an immature state in the newborn; its development is completed only by 12-13 years. According to Gundobin the lymph nodes of the newborn are soft, and their dimensions range from a pinhead to a pea. They have wide sinuses and are distinguished by an abundance of lymphatic tissue and blood vessels.

The lymph nodes are the sites of lymphocyte formation and accumulation of destroyed blood cells. Moreover, they are also a barrier blocking the propagation of infections; this function is insufficient in the newborn and in infants, improving only towards the end of the first year.

The dimensions of children's lymph nodes increase during many infectious diseases, both acute and chronic, and also in diseases of the skin and mucous membranes.

Several distinct groups of lymph nodes are described according to their location.

The following table shows the source of lymph delivered to each group of nodes.

Anterior cervical (in front of the sternocleidomastoid-pectoralis muscle)	The facial skin, the parotid lymph gland, the mucosa of the nose, throat and mouth
Posterior cervical	The skin of the neck and mucosa of the nasopharynx, and partly of the throat and mouth
Submaxillary	The oral and pharyngeal mucosa
Submental	The skin of the chin, and the upper and lower lips
Supraclavicular	The skin of the upper part of the chest, the pleura and the apices of the lungs
Mastoid process	The skin surrounding the ears, the middle ear, the skin of the external ears and the external auditory canal
Occipital	The scalp and the back of the neck
Axillary	The skin of the upper extremities, except the 3rd, 4th, and 5th fingers and the inner edge of the wrist and forearm
Cubital	The skin of the 3rd, 4th, and 5th fingers and the inner edge of the wrist and the pectoral glands
Inguinal	The skin of the lower extremities, the lower part of the abdomen, the buttocks, the perineum, the genitals, and the anus

Lesions of the lymph nodes are often observed in children, attendant on many acute and chronic diseases: the enlargement of the occipital lymph nodes in rubella (enlargement of the superficial cervical and posterior auricular lymph nodes), the enlargement of regional lymph nodes in the serum sickness depending on the point of introduction of the serum, small hard cervical lymph nodes in tuberculosis ("like little stones", according to Kisel). In healthy children not all the above-listed groups of lymph nodes are palpable; usually the anterior and posterior cervical nodes are palpated as well as the axillary and the inguinal nodes, all the rest only being palpated during corresponding disorders of the skin or mucous membranes that are the source of their lymph supply, or in certain general diseases.

When examining a child it is necessary to note the dimensions, consistency, mobility, and tenderness of the lymph nodes.

The tonsils of the newborn are small, increasing only at the end of the first year. The third, or pharyngeal tonsil is sometimes observed to grow excessively in infancy, greatly impeding respiration; at a more advanced age this growth (adenoids) is encountered more frequently, at times calling for surgery.

The *spleen* of the newborn is very small and is not palpated under the ribs. It is approximately the size of the child's palm. In a newborn child it weighs 10 g, in a year-old child 52 g, at ten its weight is 78 g; thus we see that the spleen grows intensively in the first year of life.

The Urogenital Organs

The urogenital tract consists of an excretory system (the kidneys, ureters, and bladder), and of the external and internal reproductive organs (genitals or sexual organs). Up to the age of two years the *kidneys* retain their embryonic lobulation. The renal pelvises are fully developed, the ureters S-curved. The *bladder* of the newborn has a 40-50 ml capacity. Urination occurs 16 and more times in 24 hours. In many cases it is possible to train a baby to urinate voluntarily over a potty by six months. During the first days of life the urine contains albumin (physiologic albuminuria) and a great amount of urates that sometimes appear on the diapers as reddish grains of sand; the painful passing of these grains makes the child cry loudly. The urates are precipitated in the renal tubules owing to a high concentration of urine, and are washed out with the latter (uric acid infarct).

The weight of the kidneys in the newborn is relatively higher than in adults: the kidney of the newborn weighs 10 to 12 g (1/100 of body weight), while the 150 g of adult kidney weight constitute only 1/220 of body weight.

By five to six months kidney weight doubles, increasing tenfold by the age of 15.

In the first 3-4 days of life the daily excretion of urine is slight; sometimes the baby does not urinate at all in the first 24 hours. Later, during the first months of life, the daily amount of urine excreted by the kidneys is about 300 ml, from 6 to 12 months it is 600 ml, by 3 years 800 ml, from 5 to 6—1,000 ml, at 10 years 1,500 ml, and from 13 to 15 years 1,500 to 1,800 ml. To determine the daily amount of urine passed by any child older than one year the following formula is used:

$$M=600+100(n-1),$$

where *M* is the daily amount of urine excreted at a given age, 600 is the daily amount of urine passed by a one-year-old child, and *n* is the age of the child.

For instance, the daily amount of urine passed by a six-year-old child should be $600+(100 \times 5)=1,100$ ml.

More urine is excreted in the cold time of the year than in the hot season, when a great deal of water is evaporated through the skin.

The central nervous system and its most highly organised section, the cerebral cortex, are the principal regulators of the function of the kidneys. It has been proved that excitation of the cerebral cortex causes increased urination, while inhibition leads to a decrease in urine discharge. The cerebral cortex affects the excretory function of the kidney both directly and through the hormones secreted by the posterior lobe of the pituitary body (inhibition of urine discharge) and the hormones of the anterior lobe of the pituitary (stimulation of urine discharge). The thyroid and adrenal hormones also affect urination, the first increasing it, the second decreasing it.

In their function as excretory organs the kidneys eliminate from the body various metabolic waste substances such as uric acid, urea, salts, water.

The reproductive organs. The testes of newborn male infants have already descended to the scrotum; they grow very slowly during childhood and increase rapidly at puberty. According to Gundobin, the testes are capable of producing spermatazoids by the age of 15. The foreskin (the prepuce) is comparatively long, and up to 12 months it is joined to the glans penis; the orifice of the foreskin is narrow (physiological phimosis). However, towards the end of the first year this orifice gradually widens. The ovaries of newborn female infants are almost completely developed; they grow very slowly throughout childhood and increase rapidly at puberty. The labia of the external genital organs are closed; in the first few days there may be some white discharge, due to desquamation (desquamatio neonatorum).

Sexual crises. On the second or third day of life a swelling of the breasts accompanied by a discharge of colostrum may be noticed quite often in newborn infants of both sexes. This condition is due to the hormones of the placenta and of the mother's ovaries and pituitary gland absorbed prenatally by the fetus. This engorgement calls for no medical treatment. However, if pyogenic microbes penetrate into the infant's mammary gland through its nipple, the gland becomes inflamed (mastitis), or even abscesses may be formed; these abscesses should be incised in a radial direction, as distant as possible from the nipple, so that the milk (lactiferous) ducts in girls are not cut. A less frequent occurrence is vaginal hemorrhage in newborn baby girls (resembling menstrual discharge); this condition disappears after a few days. Edema of the genitals is observed rarely.

The Endocrine Glands

The endocrines are ductless glands secreting hormonal substances into the blood stream. Similar to all other bodily organs, their functions are governed by the central nervous system through the cerebral cortex. However, the central nervous system is in its turn influenced by the hormones the endocrines secrete; often the development of the central nervous system and of the higher nervous activity of the child is influenced by pathological conditions of the endocrines. The connections and interrelationships of the ductless glands and of the central nervous system are very complex and much has still to be learned in this field. Hormones are chemical substances formed and secreted by the endocrine glands. They are delivered directly into the blood stream, and thus are transmitted to the various organs, on which their effect is very profound. The hormone of a particular gland is very specific, but the endocrines do not function in isolation from each other; the secretion into the blood of the hormone of one gland depends on the state of the other endocrines, and on the regulating influence of the central nervous system.

Any pathological process occurring in one particular gland evokes changes in the functions of the other ductless glands. The gland that is most affected determines the clinical pattern and the nature of an endocrinous disease.

In the first few months of life the endocrines are still undeveloped, secreting a low amount of hormones; however, this insufficiency in children of such an early age does not normally evoke any clinical manifestations of pathological processes due to specific endocrinic disorders. That this is so is due to the child having at birth a store of hormones received from his mother during fetal life. As the hormones are used up clinical manifestations of endocrinic hypofunction become apparent in those infants in whom the secretory functions of any endocrine gland are insufficient.

The *thymus* is a glandular organ situated in the anterior superior mediastinum; during the first 6-8 years of life it is divided into a large number of lobules. At puberty its tissue undergoes fatty metamorphosis and atrophy, and is replaced by connective tissue. It is held that this gland plays an important part in the growth of the child.

The *thyroid* gland is situated on the trachea over the 3rd-6th tracheal cartilages. It consists of two lobes connected by an isthmus; the lobes are formed of glandular vesicles lined with cuboidal epithelium; in the newborn the thyroid does not produce

any of the thyroid colloid that contains the thyroid hormone; the formation of this colloid begins slowly and gradually in infancy, the maximum rate of formation being attained at two years. Intensification in the activity of the gland is also observed at puberty (enlargement of the thyroid in girls). The function of this gland is the regulation of metabolism. A congenital absence of the thyroid gland or its atrophy are the cause of a severe disease, myxedema.

The *parathyroids* are located laterally on the thyroid, two on each side; they consist of columnar epithelium (from which the name "epithelial bodies" comes). The parathyroids are very important in renal phosphorus excretion and (principally) in upholding the level of blood calcium; this latter consideration has led many authors to ascribe an important role in the etiology of spasmodophilia to the parathyroids. Their surgical removal leads to tetany.

The *pancreas* is a dual gland, possessing both external and internal secretion. The so-called islets or islands of Langerhans, or simply the islets of the pancreas, secrete a hormone called insulin that regulates carbohydrate metabolism, keeping blood sugar at its proper level. Dysfunctions of the pancreas cause diabetes.

The *suprarenal or adrenal* glands are quite large in the newborn, consisting almost entirely of cortex, the medulla developing only by the age of two. The chief hormone of the adrenal medulla is called epinephrine (adrenalin); it is a vasoconstricting substance that stimulates the sympathetic nervous system and regulates the formation of pigment. Acute failure of the adrenals causes collapse, and even death. Diseases of the adrenals (tuberculosis, for instance) evoke the development of Addison's disease.

The *pituitary* (the hypophysis cerebri) is a gland lying in the sella turcica of the sphenoid bone of the skull and consists of anterior, intermediate and posterior lobes. The anterior lobe secretes a principle that regulates growth; hyperfunction of this lobe leads to gigantism, its hypofunction to dwarfism. The hormone secreted by the intermediate, and partly by the posterior pituitaries regulates metabolism; hyposecretion of this hormone leads to adiposogenital dystrophy—obesity and retarded development of the reproductive organs, and also to diabetes insipidus.

The Digestive Organs

The morphologic and functional development of the digestive organs of the child are correlated with the development of the central nervous system and the principal regulator of all vital processes, the cerebral cortex.

All the digestive organs are interdependent in their general function of digesting food, and they are directly affected by the central nervous system. The digestive (or alimentary) tract is very active in childhood, as the continuous growth and development of the child call for a greater quantity of nutritive substances as compared with adults.

The digestive system of the newborn infant is immature; the most easily digested food is breast milk, a substance that supplies the infant with all the nutritional components (proteins, fats and carbohydrates) needed for its growth and development. As time passes and the digestive organs become more developed and differentiated, the child can digest and assimilate coarser food.

It is also important that the baby be fed at regular hours; a conditioned reflex to time is thus established, and as feeding time approaches the primary digestive juices are secreted; by then the stomach will already have been emptied of previously taken food.

The body requires a constant supply of food; however, nutritive substances cannot be assimilated by the organism in the form they have been ingested. Only water, mineral salts, and vitamins are assimilated in their initial form, all other substances (proteins, fats and carbohydrates) must go through a complex process of digestion: first they are converted from a solid to a semiliquid state, and then subjected to chemical transformations. These chemical reactions are effected by special substances, enzymes, contained in the digestive juices of the stomach and intestine.

The sucking and swallowing (deglutitory) instinct is fully developed in the newborn. The lips grasp the nipple firmly, the jaws compress it, the tongue is drawn back, the lower jaw descends; negative pressure causes the milk to pass into the child's mouth after several sucking motions, and to be swallowed. The salivary glands barely function in the first three months; this causes dryness of the oral mucosa. The stomach lies horizontally, and has an unstable irregular shape. The pylorus (the circular opening of the stomach into the duodenum) closes tightly, but the cardia (the esophageal orifice of the stomach) does not shut so tightly: this is why infants frequently regurgitate their food. The volumetric capacity of the stomach in the newborn is 100-150 ml of liquid; by six months it increases to 200-300 ml, and by one year attains 400-500 ml. The digestive juices of children include the same components as in adults (chymosin or rennin, hydrochloric acid, pepsin, lipase). The hydrochloric acid and chymosin content of the gastric juice of the newborn is relatively lower than in adults,

increasing gradually as the child gets older. The pancreas is fully developed and functions as in adults; it grows very rapidly (from 2.5 g at birth to 50 g at puberty). The liver is large, its lower edge protruding 2 cm beneath the ribs. By the end of the first year it lies on the level of the lower edge of the ribs. The amount of bile secreted is much less than in adults. In children the intestine is comparatively longer than in adults.

Intestinal flora. The alimentary tract of the fetus is sterile; usually no microbes are to be found in the meconium (the first fecal discharge of the newborn). However, the first breath of air brings with it into the stomach and intestine a swarm of the most various bacteria; they are already abundant in the feces on the second and third day after birth. The majority of these bacteria are located in the lower reaches of the small intestine and in the large intestine. When a normal breast-feeding routine has been established an anaerobic organism appears in the intestine and feces of breast-fed infants; it is called *Bact. bifidus*, or *Lactobacillus bifidus* (from its forked ends in cultures); these bacteria produce lactic acid by hydrolysing lactose (milk sugar), causing the reaction of the fecal matter to become acid. The majority of bacteria require a weak alkaline reaction for their development; an acid reaction of the feces causes the other bacteria to disappear. *Bact. bifidus* constitutes approximately 90 per cent of the entire intestinal flora. Hence intestinal disorders are observed less often in breast-fed infants. When babies are nursed on cow's milk conditions for the development of *Bact. bifidus* are evidently unfavourable, and its place is taken by *Bact. coli* (or *Escherichia coli*), the intestinal rod; in this case the reaction of the feces is weakly alkaline, and conditions become favourable for the development of various bacteria.

The *meconium* (first fecal discharge) is a greenish-black pasty mass, consisting of the amniotic fluid swallowed by the fetus, mixed with mucus, desquamated epithelial cells, bile, secretion of the intestinal glands, lanugo.

On the third or fourth day the feces of breast-fed infants, containing undigested food, numerous bacteria and glandular discharge, becomes a golden yellow and emits a sourish odour (owing to the formation of lactic acid). If the child is being nursed on cow's milk the large amount of casein in it makes the feces lighter in colour and more solid, with a slightly putrid smell (decaying proteins). However, it must be remembered that 35 per cent of healthy, normally developing breast-fed children may have greenish stools (physiological dyspepsia).

The *digestion of the infant* depends on his food. Human milk has a high albumin and low casein content (albuminous milk); it is digested in two and a half hours, much faster than cow's milk with its high casein level (casein milk) that takes three to three and a half hours to digest. In the mouth the milk is mixed with saliva; after the third month of life a diastatic enzyme (ptyalin) appears in the saliva; this enzyme possesses the property of hydrolysing starch to dextrin (when babies are bottle-fed on formulas).

In the stomach the milk is curdled by a milk-coagulating enzyme, rennin, contained in the gastric juice; cow's milk curdles rapidly, forming coarse flakes (calcium caseinate or curds), breast milk curdles slowly, forming small flakes. The whey, with a part of the proteins and the greater part of fats, is delivered into the duodenum, while the calcium caseinate is hydrolysed by hydrochloric acid and pepsin in the stomach. Owing to its high protein content cow's milk requires much more hydrochloric acid than human milk does. The products of hydrolysis of milk proteins are peptones; in the intestine the pancreatic juice, bile and glandular secretion continue the hydrolytic process; fats are emulsified and split by bile and lipase, while sugar (lactose) is hydrolysed by various enzymes (lactase and others). The semiliquid product of digestion, the chyme, is absorbed by the small intestine, while the roughage (the undigested food residues) passes into the large intestine; here the water is absorbed from it and the fecalia are solidified. About 95 per cent of the constituents of milk are assimilated in the intestine.

The importance of food for the child. Proteins are the plastic material of which the cells of the rapidly growing organism are built. Fats are partly stored in the subdermal tissue, and are partly used up together with the carbohydrates to produce warmth and muscular strength (transformation into energy). Water is also retained in the body under the influence of carbohydrates and salts (sodium chloride); water is very necessary for the normal activity of all the tissues. Milk contains calcium and phosphorus, elements required for the proper growth of the bones and for the activity of the central nervous system. The tissues of the infant's body contain a lot of water, and lose water very rapidly; therefore, these stores must be replenished regularly, particularly in the summer, and babies should be given water between nursing times.

In addition to proteins, fats, carbohydrates and water the growing organism also requires vitamins.

In the child, as in the adult, food is the source of muscular energy and warmth and of material for forming new cells in place

of atrophied cells; moreover, about one-third of the entire food ingested is used up in the formation of new tissues. In the first three months of life babies need 120 calories per kg of body weight a day, in the second quarter 110, in the third 100 and in the fourth 90 calories. Later the number of calories required daily decreases, going down to 50 cal/kg at puberty. One g of protein or carbohydrate produces 4.1 calories, while 1 g of fat produces 9.3 calories (a calorie is the amount of heat required to raise the temperature of 1 kg of water by 1° C). The amount of calories required at different ages: 400 at one month, 700 at six months, 800 at one year, 1,300 at six years, and about 2,400 calories at puberty.

The liver. The liver plays an important part in digestion and metabolism. Its principal function is the secretion of bile; bile activates all enzymes, particularly the fat-splitting enzyme lipase (15 to 20 times). The bile emulsifies the fats, i.e., breaks them up into minute globules, multiplying the surface of the nutritional fat many times over and thus facilitating the action of the lipase.

Moreover, the liver is a barrier that detains the toxic products formed in the intestine in the process of digestion; the liver cells render these products harmless, converting them into nontoxic substances that are subsequently carried by the blood stream into the kidneys and excreted from the body.

The liver of the newborn child is comparatively large; its lower edge protrudes from the subcostal area by 2 cm; by the end of the first year it is level with the lower ribs. Up to the age of 5-6 years the liver is still normally palpated, but any enlargement of the liver and its palpation in the abdominal cavity at a later age is pathological.

The amount of bile secreted at birth and in the first months of life is small; the development of the liver cells is not completed until the age of five or six years.

The Sense Organs

The sense organs are a part of the united system of analysers. Knowledge of this system was greatly expanded in the teachings of Pavlov.

Pavlov divided the system of analysers into a peripheral or receptive part, a conducting part, and a central part in the cortex, considering them all as one system. According to his teachings the sense organs belong to the peripheral part (receptors); they respond to external stimuli and transmit these stimuli through the

conducting part to the central part—the cerebral cortex. In the cerebral cortex the impulses are minutely analysed and synthesised, and perceived as sensations (hearing, vision, taste).

Hearing. The auditory organ is poorly developed in the newborn; sound is conducted mainly through the bone tissue, as at birth the tympanic cavity is still filled with amniotic fluid, a mucinous liquid; the latter obstructs the conduction of sounds. The liquid is gradually resolved and replaced by air; this process is attended by a gradual development of the acuity of hearing. The newborn child reacts only to loud sounds by starting; at four months a child turns its head in the direction of loud sounds, after one year its hearing is well developed. The external auditory canal is very narrow in the newborn, it consists of cartilage, not bone. The space between the upper and lower walls of the auditory canal is only a narrow slit in the newborn; this accounts for the detention of suppurative discharge in ear diseases in infancy. Another specific feature of babies in the first few months of life is that the auditory (eustachian) tube that opens into the nasopharynx at one end, is short and wide; this facilitates the penetration of infections from the nasopharynx to the middle ear.

Vision. A newborn child sees from the first days of life. Its orbits are relatively large. The curvature of the cornea is more pronounced and the lens more bulging than in adults. The eye of the newborn is hypermetropic (far-sighted), accommodation is weak, therefore, no clear image is obtained on the retina. Up to 2-3 weeks the movements of the eyeballs are not co-ordinated, there is likewise no co-ordination between the movement of the eyelids and the eyeballs. Only after two months does the child fix objects with his eyes and follow them; after 4-5 months he looks at things consciously. In the second half of the first year babies can discern colours, and at three years they give them their correct names.

The newborn infant cries without tears, only after two months do tears begin to flow. The nasolacrimal duct is comparatively wide in the first months of life, causing frequent irritation of the conjunctiva and the permeation of infection from the nasal cavity.

The olfactory sense (smell). This sense is very weakly developed at birth; however, sharp odours (ammonia, vinegar) are perceived as being unpleasant from the very first month of life. So narrow are the nasal passages that the slightest swelling of the mucosa obstructs them, impeding respiration.

In the second year of life children can distinguish various odours quite well (pleasant and unpleasant ones).

The gustatory sense (taste). This is a sense that is well developed from the very first days of life; infants distinguish sweet, bitter and sour food. By the age of four or five months the sense of taste becomes still better. Disturbances of the gustatory sense attend certain diseases; thus, taste reflexes are greatly impaired by rickets.

The tactile sense (touch). Infants are born with the sense of touch; when their lips are touched they react with sucking movements. Their palms, soles, and facial skin also possess tactile sensitivity.

In the first weeks of life the pain threshold is low; however, a prick of the nose or mouth on the second day of life will evoke a grimace from the baby.

The temperature sense is well developed in the newborn: the new and colder environment the infant emerges into upon birth causes it to cry, tremble and become pale; on a heated pad it calms down and its skin becomes rosy-pink.

Motor and Mental Development

Motor development in healthy children is directly associated with the development of the central nervous system—the cerebral cortex, and is to a large extent connected with the child's environment and training. Retarded mental abilities in some children may be due to deficiencies in the development of the central nervous system and to diseases of this system both in fetal and postnatal life.

However, even in such cases a proper regimen and timely medical treatment may greatly alleviate the pathological deviations in the development of the child's central nervous system. A healthy *newborn infant* is helpless, its movements are irregular, it cannot change the position of its body. The muscles of the newborn are, as it were, constrained (physiological hypertonia of the muscles). The movements of the newborn child's eyes are uncoordinated; often newborn infants display squints that subsequently (by 1-1½ months) disappear, and the baby begins to fix its gaze on bright objects.

At *one month of age* the baby starts at sudden sounds, smiles, begins making indefinite sounds, attempts to lift its head.

At *two months* the baby already holds its head, follows moving objects with its eyes, smiles when it is prodded and when people smile and talk to it.

In its *third month* the baby turns its head at a sound, lifts its head and the upper part of its body when lying prone, supporting itself on its elbows. Lying on its back it turns over on a side.

The movement of the baby's hands become freer and more conscious: it reaches for toys, takes things into its mouth. It opens its mouth when it sees its mother's breast or its nursing bottle. The sounds the baby emits become more varied. When in a good mood it laughs, and cries when dissatisfied.

By *four months* the physiological muscular hypertonia in the lower extremities disappears; his movements become freer; lying on its stomach, the baby can raise itself by supporting itself on its hands; it holds things securely. It starts making melodious and guttural sounds, frets and cries when dissatisfied with something.

At *five months* the baby already knows its mother well, and discerns strangers. It sits well, holding on to something, but its back is still curved and not very strong. When held under the armpits the child stands up straight; it grasps things, holds them in its hands and pulls them into its mouth.

A *six-month baby* turns over from back to stomach, sits without help, attempts to crawl; it makes steps when held up. At this period it begins pronouncing syllables: "ba", "ma".

Seven-month babies begin crawling on all fours independently, get to their knees in bed, holding on to the bars or net of the bed or play-pen. They stretch out their arms to their mothers or people they know well, and repeat the syllables they know several times in a row: "ma-ma-ma", "ba-ba-ba", and so forth.

At *eight months* the baby sits up alone, gets to its feet holding on to the bed-rails. Its face expresses interest at the sight of new toys and objects. It attempts to walk, holding somebody's hand.

By *nine months* babies walk holding on to a walker or held by both hands; they start picking up tiny objects and follow thrown objects with their eyes.

At *ten months* the baby utters its first simple words "mama", "baba"; it reacts to kind and angry tones of voice; gives monosyllabic names to toy animals: "Moo"—cow, "ow"—dog.

By the end of the *first year of life* (11-12 months) the child begins walking alone, knows the names of many things, points to various parts of his body when asked "where is your nose?", "where are your eyes?", "where are your ears?". He bends down to lift things, places one object (toy blocks) on another (Figs. 9 and 10).



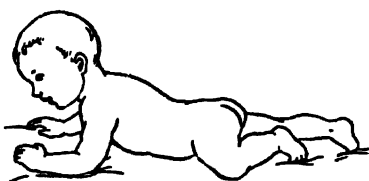
Newborn



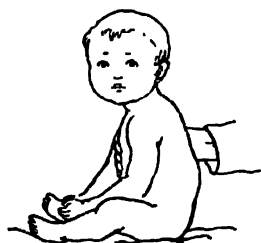
1 month



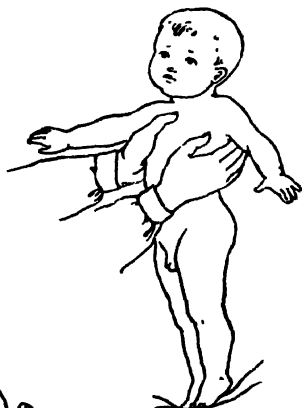
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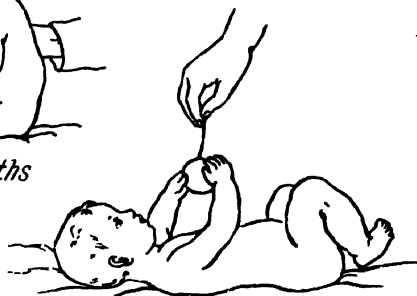
3 months



4 months



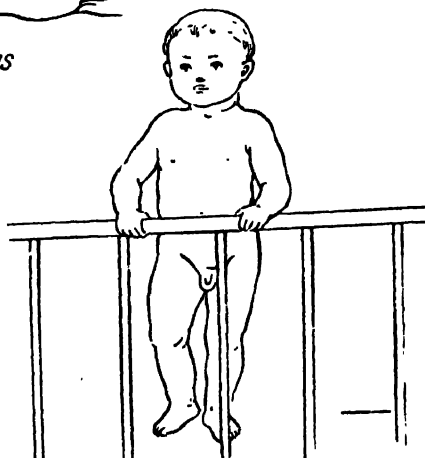
5 months



6 months



7 months



8 months

Fig. 9. Development of motorium

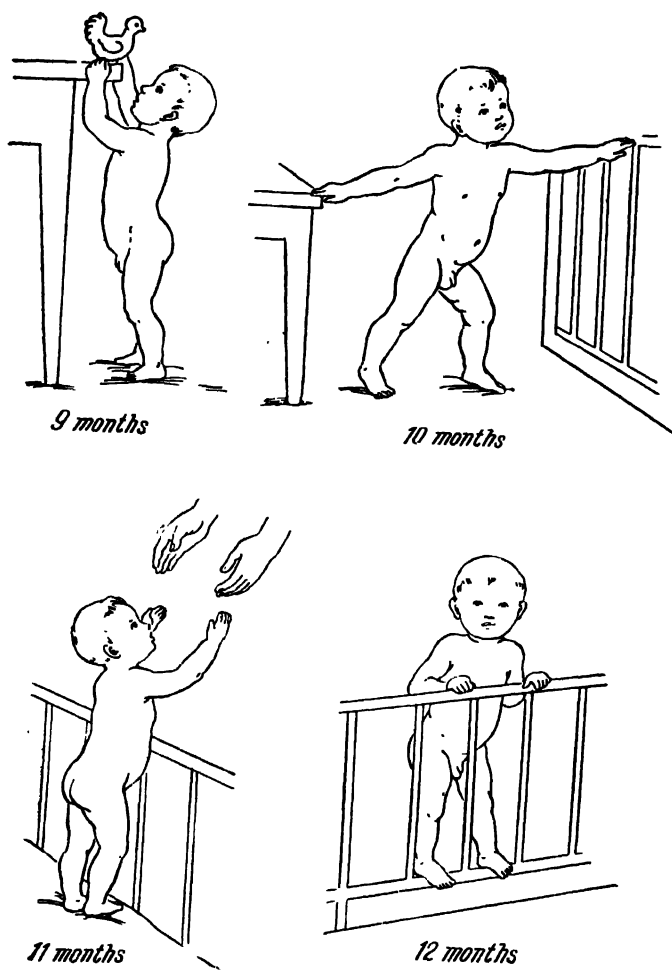


Fig. 10. Development of motorium

In the *second year* of his life the child walks confidently; his vocabulary increases; he looks at pictures, is interested in the society of other children, begins saying two-three words in a row, plays with a ball, becomes bladder- and bowel-trained.

Between the ages of two and three the child begins to recognise objects in pictures, asks many questions, memorises simple verses and tunes. His vocabulary grows to 250-300 words. At this age some traits of character are exhibited: pity, vanity, and so forth.

The mental development of the child progresses still further

between three and four years. He becomes inquisitive, memorises verses easily; at this age children try to draw human figures.

In the *preschool age* (from four to seven) traits of the child's personality become clearer, his mental development advances considerably. He becomes more and more in need of the company of other children, of playing games with them; after 6-7 years children begin reading, some even write.

The subsequent period of the child's life is spent at *school*; this is a stage full of diverse interests and needs. The personality of the child is formed at this time, and his intelligence receives further development.

The above data on the gradual development of the intelligence and motor faculties of the child are only approximate; parents and child care personnel should be acquainted with them in order to be able to facilitate the child's subsequent nervous and mental development by proper care.

Unfavourable family conditions, standard routine and the underestimation of age group specificities when children are brought up collectively, indifference to the interests of the child—all this may not only exert an unfavourable influence on physical development, but may likewise have an unfavourable effect on mental development and abilities, on the formation of a child's personality.

Sleep

We know from the teachings of the famous Russian physiologist Pavlov that sleep is one of the forms of internal inhibition in the cerebral cortex.

The cells of the child's cerebral cortex receive many various kinds of stimuli during the day; their faculty of normal function becomes exhausted and further stress may lead to the destruction of these cells. To avoid destruction the cortical cells pass into a state of inhibition—protective inhibition. In this state the cells are at rest, and their efficiency is restored; high efficiency of the cortical cells guarantees a healthy state of the body as a whole. Protective inhibition of the cortical cells, that is, sleep, prevents their overexertion and resultant destruction.

Upon passing into a state of inhibition the cortical cells of the brain transmit this condition by induction to the lower lying sections of the brain (the subcortical area), and deep sleep ensues. During sleep the entire activity of the various analysers ceases (vision, hearing, motor functions). The younger the child, the more sleep he needs.

The newborn baby sleeps practically all the time, waking only to feed. This protects the young cells of the cerebral cortex from excessive stimulation caused by external factors. As the child grows older and begins to have more and more relations with his environment the cortical cells of his brain become more differentiated and are capable of sustaining longer and deeper excitation, so that the child's need for prolonged sleep decreases.

The following sleep norms are considered most appropriate for the different age levels; the physical development and general health of the child may require a corresponding increase in these norms (Table 5).

Table 5

Hours of Sleep Required by Children at Various Age Levels

Age (in years)	Hours of sleep per day	
First months of life	20-22	Two periods of sleep in the daytime
1	16-17	
2-3	14-16	
4-5	13-14	One nap in the daytime
6-7	12-13	
8-10	11-12	
11-12	10-12	A nap in the middle of the day according to medical indications
13-14	9.5-10	
15-17	9-9.5	

The child should be given complete rest during his sleep—silence in the room, dimmed light.

Naps are healthier when taken in the fresh air, as sleep is then deeper and longer. During the night the air must not be close; the room should be well aired before the child is put to bed.

In children's therapeutic and prophylactic institutions (hospitals, crèches, nursery schools) good care must be taken that the children get the amount of sleep prescribed for their respective ages, both at night and in the daytime. Sick and weak children must sleep longer. Recuperation is slower if sleep is insufficient.

A strict regimen of sleep must be adhered to both for healthy and sick children: no loud talking is to be permitted, nor cleaning up, taking temperatures of sleeping children, administration of medicines in the early morning or the conduction of various procedures that might disturb the child's sleep.

NUTRITION

Feeding the Healthy Child

The child differs from the adult in that he is growing and developing, that is, the mass of his body is on the constant increase, he grows in length, his internal organs develop and their functions improve. To ensure the normal progress of these processes the child must constantly receive *nutritive* substances from external sources; these substances provide material for *building the cells* of the growing organism and *making up its losses*, and they are the *source of warmth and energy* required for the functioning of the human body and its activity (movement, digestion, heartbeat, mental processes). These nutritive substances are the child's food. Similar to adult food, it consists of water, proteins, fats, carbohydrates, salts, vitamins. These substances are all constituents, in various proportions, of the food of human beings. The nutritive substances of the child's food should satisfy his growth requirements and afford a sufficient amount of energy (warmth) for his bodily activity. Normal development calls for food containing all the elements comprising the child's body. It has been estimated that the adult body contains approximately 60 per cent water, 14 per cent proteins, 20 per cent fats, 1 per cent carbohydrates and 5 per cent mineral substances.

The tissues of young children, and particularly of newborn infants, have a much higher water content—up to 75 per cent, only 25 per cent comprising the solid residue.

The growth and development of the child's body is accompanied by a decrease in the water content, and a corresponding increase in solids.

Proteins are the basis of all living cells. They include nitrogen, and are the plastic material which serves for building up the tissues; they are also a partial source of energy. There is nothing that can substitute proteins as plastic materials in the diet of the child.

Proteins occur in products of both animal and plant origin; however, the human organism does not digest all proteins with equal ease; this is particularly true of babies.

There are full-value and inferior proteins; the first are of animal origin (milk, meat, eggs), containing all the amino acids required for building up the cells of the body, their structure approaching that of the proteins of the human body.

Proteins of plant origin (cereals, bread, legumes, vegetables) contain only some of the amino acids necessary to the human body

and thus do not provide full-value nutrition, particularly for the growing child.

Adult food contains both animal and plant proteins, but the growing child needs a greater amount of animal proteins than adults do. This pertains particularly to very young children; the younger the child, the higher must be the quality of the proteins his food contains.

A breast-fed child gets 2-2.5 g of protein per kilogram of weight; this is quite sufficient, as human milk is the most valuable infant food and is digested much easier than any other. But if infants are bottle-fed on cow's milk mixtures they need up to 4 or 5 g of proteins per kilogram of weight. However, excessive amounts of protein above established norms lead to undesirable results: appetite deteriorates, dyspeptic symptoms may appear, the elimination of nitrogen with the feces and urine increases; this may finally lead to a lower retention of nitrogen in the organism, causing growth and developmental disturbances.

Table 6

Fat Requirements per Kilogram of Body Weight

Age of child	Fats (g) per kg of weight
Breast-fed infant	5.5-6.0
Bottle-fed infant	4.5-5.0
Child at the age of (years):	
1-4	4.0-3.5
4-7	2.0-2.5
over 7	2.5-2.0

Fats are the source of energy in the body; upon oxidation they eliminate a great amount of heat. Besides this, fats must be stored in the organs and tissues. Suffice it to say that there is not an organ in the human body that does not contain fat. Fats are the carriers of vitamins A, D, E, and K.

Lipoids (or lipids) are a group of fat-like substances the molecules of which contain phosphorus. Their high metabolic activity makes them very important in all vital processes, as they are activators of intracellular processes.

The most valuable of the lipids are the *lecithins*, substances playing an important part in the normal function of the nervous system. Lecithins occur in milk, butter, egg yolk, fish roe, and brain. However, not all the fats encountered in nature can be digested by the child's organism with equal ease. Best of all in this respect

is human milk; breast-fed infants digest up to 90-95 per cent of the fat contained in human milk. 80-85 per cent of cow's milk fat is digested. Fats of animal origin—butter—are the most valuable fats for feeding children, particularly babies after they have been weaned. Inferior animal fats are drawn butter, margarin, suet and pork fat. Still lower in this respect are vegetable fats (sunflower-seed, cotton-seed, and other vegetable oils).

The younger the child is the more animal fats his food should contain. Children under three years should be given only fresh butter in their food. After three years other animal fats may be added to butter—drawn butter, margarin, lard, and also a small amount of vegetable oils. The younger the child the more fats he requires. To ensure proper digestion of fats the child's diet must include carbohydrates—two to three times more than fats.

Carbohydrates are the chief source of bodily energy and warmth. They include a number of starchy and sugary substances. Carbohydrates are highly important in the child's nutrition, as they economise proteins, retarding their destruction in the organism for the formation of energy, thus facilitating a fuller utilisation of proteins in plastic processes.

The carbohydrate content is high in grain products, cereals, vegetables, berries and fruits. The most easily and swiftly digested carbohydrate is sugar; taken with food after prolonged physical exertions, or when a person is fatigued, it soon restores bodily strength.

Breast-fed infants receive 10 to 12 g of carbohydrates per kilogram of their weight; in artificial feeding the amount of carbohydrates increases somewhat—12 to 14 g/kg. Children over a year old require 12 to 15 g/kg of carbohydrates.

From the intestine the carbohydrates are assimilated into the blood in the form of glucose. Superfluous glucose is deposited in the liver where it is synthesised into animal starch—*glycogen*.

Stored in the liver, glycogen is converted, as the system requires (muscular exertion), into sugar (glucose); the glucose is taken up by the blood stream and conveyed to the organs that need it. Besides being stored in the liver glycogen is also contained in the muscles, but in a much smaller amount. Excessive deposits of glycogen in the liver and muscles are in some rare cases displayed as a specific disease, the glycogen disease (glycogenosis). On the other hand, an excessive amount of glucose in the blood evokes the clinical symptoms of diabetes melitus.

Mineral substances. Aside from proteins, fats and carbohydrates human food must also contain inorganic, or mineral substances:

calcium, sodium, potassium, phosphorus, iron, copper, iodine, sulfur, and others, all necessary constituents of the body cells and tissues, some of them are important components of the bones. All these substances are diffused in the system in a dissolved state. Mineral salts are highly active in water metabolism, and affect protein and fat metabolism.

The salts most important in the child's diet are calcium, phosphorus and iron. *Calcium* salts are necessary for building up the bones; the required amount per day is 1 g. Foods with the highest calcium content are milk, cheese, egg yolk, beans, cauliflower and some other products.

Phosphorus is a constituent of nervous tissue and of other cells. It is also needed for the formation of bone tissue. The body needs 1.5 to 2 g per day. Phosphorus occurs in milk, egg yolk, liver, meat and legumes, in millet and other grains.

Iron is one of chief constituents of hemoglobin, the respiratory pigment of the blood that carries oxygen to the tissues. Fifteen mg of iron are required daily. Foods rich in iron are egg yolk, meat, liver, kidneys, coarsely milled rye and wheat bread.

Sodium and potassium occur in the organism in the form of chlorides, bicarbonates and phosphates. These compounds are utilised by the system for neutralising the acid products of metabolism. Sodium chloride (common or table salt) is necessary for the formation of hydrochloric acid in the stomach; this acid plays an important part in digestion. The average requirement of the human system in this salt is 10-15 g a day. Strenuous muscular exertions, running, walking, and excessive perspiration increase the requirement in salt, and additional salt is then needed at meals.

Such mineral substances as iodine, sulfur, copper, magnesium, and a number of others are needed in very small amounts; however, even in very insignificant amounts, measured in thousandths, ten thousandths, or even lesser parts of a gram, these substances are extremely important factors in metabolism and the vital activity of the organism.

The food and water taken by adults and children usually contain all the necessary mineral substances in sufficient amounts. In localities where the water is deficient in iodine a small amount of this element has to be added to the food, otherwise the functions of the thyroid gland become disturbed (deficient secretion of its hormone), and a disease of the gland—endemic goitre—ensues. Mental development is significantly retarded by this disease, and it leads to a particular kind of idiocy. All the mineral substances

needed by the child are sufficiently contained in milk, eggs, meat, vegetables and fruit.

Water. As we have already mentioned, in the newborn stage the tissues have a much higher water content than in adulthood, and therefore babies require comparatively more water. Water is a constituent of all the tissues, including bone, and it is the principal component of all systemic liquids and secretions and excretions of the body. All the processes that go on in the organism are connected with water. The water requirements of man depend on age and activity.

A newborn infant should get 150-160 ml of water per kg of body weight; at six months the water requirement decreases to 120 ml, by one year—to 100 ml, by two—to 90 ml, and at the age of 12 or 13 years it goes down to 40-45 ml/kg.

About one-sixth of the entire liquid needed by the system is supplied by liquid and partly by solid foods; the remainder comes as drinking water, tea and other beverages. Intensified muscular activity (running, fast walking) and also hot weather significantly increase the need for water. Water deficiency, particularly in early childhood, is tolerated with great difficulty.

Water deficiencies in children may be caused by intensive water depletion due to vomiting, diarrhea, excessive perspiration; such a deficiency is very harmful to the child's health. Therefore, one of the first measures to be taken in these cases is timely replenishment of water losses by drinks, normal saline solution enemas, and at times even subcutaneous or intramuscular infusions of normal saline or Ringer's solution, plus 5 per cent glucose solution (see section on dyspepsia).

Breast-Feeding

The majority of women in the Soviet Union breast-feed their children, both in cities and in the country. According to the data of Moscow infant health centres approximately 92 per cent of mothers feed their babies at the breast alone in the first few months of life, or, if they do not produce enough milk, they give a formula plus the milk they do have. This is very important, as the mortality rate among breast-fed infants is eight times less than among bottle-fed babies.

Studies made of breast milk and of the milk of various animals have shown that human milk contains more fats and lactose and much less proteins and salts than animal milk. Besides the quantitative difference there is also a difference in quality: cow's milk

has a high casein content, while human milk contains much more albumin. The high level of protein and mineral salts in cow's milk is necessary for the growth of calves, whereas for human babies this amount of protein is only an unnecessary burden. The amount of enzymes and vitamins in the milk of various animal species is also evidently dissimilar. A microscopic study of human milk shows that it contains fat globules of different dimensions and single leukocytes. The quality of the milk is not affected by the dimen-

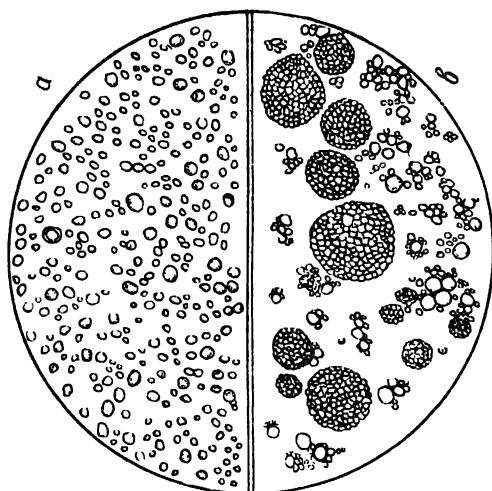


Fig. 11. Colostrum and human milk under the microscope
a—milk; b—colostrum

sions of the globules. In general it should be remembered that very seldom is bad human milk encountered, and that there is no better food for babies than breast milk. The milk of some women is low in fat; however, this deficiency is compensated by the baby withdrawing a larger amount of milk, and development is normal in such cases. Fat and protein deficiencies may be compensated to a certain extent by giving the mother supplemental nutrition.

The first 2-3 days after delivery a yellowish liquid is secreted from the breast (the colostrum); it has a high content of protein and fat globules, and a small amount of roundish cells containing drops of fat (colostrum bodies) (Fig. 11). Every mother can and should breast-feed her baby. A new pregnancy is no contraindication to breast-feeding; the great majority of women can and should nurse their babies on the breast up to the sixth month of a new pregnancy.

If the mother contracts an acute disease (pneumonia, various forms of typhoid fever, puerperal sepsis, scurvy) breast-feeding may still be continued. In severe cases mixed feeding is advisable. Influenza, sore throat (tonsillitis, laryngitis) and other contagious diseases call for precautions: the mother should cover her nose and mouth with a gauze mask, put on a clean smock while nursing, and wash her hands very thoroughly in order not to infect the baby. If the mother has scarlet fever, diphtheria or erysipelas the milk should be expressed from the breast manually or with the

aid of a special pump, then sterilized and given to the baby in a bottle. A nursing mother afflicted by mastitis may give her child both the healthy and affected breasts. If the mother has TB in a closed form breast-feeding is permitted, only precautions must be observed (a handkerchief tied over mouth and nose, no kissing of the baby, etc.) In cases of open forms of tuberculosis, when the patient runs a high temperature and the tubercle bacillus is found in the sputum, breast-feeding should be stopped. Syphilitic mothers may breast-feed their infants, even if the baby shows no sign of the disease, as in such cases the child has the disease in a latent form anyhow. If the mother has contracted the disease after the birth of the child, she should not nurse it herself, but should give her sterilised milk to the baby in a bottle. Mothers afflicted with severe mental disorders are not allowed to breast-feed their children.

Impediments to breast-feeding are deep fissures on the nipples, flat and retracted nipples, engorgement (induration) of the breasts. In all these cases it is recommended to feed the child through a nipple shield. To prevent cracks the nipples should be rubbed with diluted alcohol during pregnancy, and if cracks do form penicillin ointment should be applied to them (but not to the entire nipple!), and then a bismuth salve. More recently nipple cracks have been successfully treated by irradiation under quartz mercury vapour lamps. Engorged breasts should be massaged.

Obstacles to feeding may occur in the infant itself, too. Such obstacles are premature delivery and congenital weakness, harelip and cleft palate, nostrils blocked due to a cold, thrush and stomatitis, pneumonia, severe general diseases (encephalitis, severe forms of dysentery and pyelitis), neuropathy.

Children with harelip and/or cleft palate usually do not take the breast; they are fed with manually expressed breast milk through a bottle; the rubber nipple of the bottle is pushed almost to the posterior wall of the pharynx, the child lying on his side. If general nutrition is satisfactory surgery is performed as soon as possible. During the first 3-4 days after the operation the child is spoon-fed or his milk is given through a pipette, so that the sutures do not part. One teaspoonful of a 4 per cent solution of sodium bromide is given three times a day to keep the baby quiet. A week after the sutures have been removed the child can be put to the breast, but carefully. If the sutures do part a second operation may be performed only several months later. Children under five years of age should not be operated for cleft palate.

If the baby has rhinitis his nasal passages should be cleaned with

a twisted swab of cotton (no match-stick!) dipped in vaseline (petroleum jelly) or mineral oil. Besides this, a 1 per cent solution of protargol may be used, two drops into each nostril three times a day, or a 1 : 10,000 solution of adrenalin (same dose). If dry crusts form in the nostrils a little boiled mineral oil should be instilled. In pneumonia and other severe diseases babies are given small amounts of manually expressed breast milk from a bottle, spoon, or through a tube.

A nursing mother may eat anything she likes. She should not take alcoholic beverages (beer, wine), nor smoke. It is good to eat fruits, vegetables, and to take ascorbic acid (0.3 to 0.5 per day) during pregnancy, in the lying-in period and during lactation. Plenty of fresh air and bathing in the summer are very beneficial for the nursing mother. In general, frequent ablutions of the body and breast, and hand-washing are important in preventing mastitis. The nursing mother needs eight hours of sleep.

Although it has been proved that many medicines pass into the milk, still the quantity is so insignificant that the mother may take almost any medicine the doctor finds necessary to give her (iron and arsenic preparations orally and parenterally) except narcotic drugs (opium, morphine). If the mother suffers severe pain injections of pantopon, morphine, etc. are permitted, only the mother must express her milk manually three hours after the injection, and not put the baby to the breast before six hours have passed.

To produce a sufficient amount of milk the mother must take approximately two litres of liquid a day (including milk, soup, tea, water), and must make sure that the baby empties the breast completely. There are at present no known drugs that increase lactation. Sometimes the breast is irradiated by mercury vapour lamps and 10-15 drops of vitamin A are given twice a day.

It happens at times, in neurotic mothers, that the milk trickles out of the breasts between nursing times. Besides general strengthening measures of treatment, the breasts should be lifted and firmly supported by a bandage, the nipples massaged and the breast sponged with room-temperature water and irradiated under a mercury vapour lamp. If the breast is engorged and the child a weak one, the mother should massage her breast a little before nursing and express some of the milk, and only then put the child to the breast. Neurotic women produce less milk under mental shock. The mother must be tranquilised, and the milk will come in again. Sometimes neurotic mothers complain of acute pains in the breast during nursing (mastodynia). Bromide preparations and mercury

vapour lamp irradiation (15 treatments on alternate days) give good results in some cases.

It is usually from six to twelve hours after delivery that the mother's breasts become distended and produce the yellowish milky fluid called colostrum. Therefore, the infant should be put to the breast six to twelve hours after birth, for when this is done later (on the second day, as was practised formerly) the child hungers and loses too much weight in the first week.

Newborn infants should be fed every three hours, seven times a day. A six hour break, from midnight to 6 a.m. is necessary, as the stomach needs a rest. The baby should be put on a strict schedule (6 a.m., 9 a.m., noon, 3 p.m., 6 p.m., 9 p.m. and midnight); differences should not be longer than 10-15 minutes earlier or later.

If the baby is asleep at nursing time, it should be uncovered cautiously, changed, and put to the breast. Each nursing takes, on the average, about 20-30 minutes, but it must be borne in mind that a weak infant may only get the necessary amount of milk in about thirty minutes, while a strong one can withdraw the same amount in five to ten minutes. The baby must not be allowed to sleep at the breast, it should be gently prodded and patted and encouraged to suck.

After the infant has been nursed it must not be carried around or jostled, to avoid regurgitation; it should be put to bed very carefully. When the baby is 2-3 months old it may be transferred to a three and a half hour schedule (six times a day), and at five months to a four-hour schedule (five times a day: 6 a.m., 10 a.m., 2 p.m., 10 p.m.).

A woman can produce one to two litres of milk a day on the average. But it must be remembered that the child does not take the same amount of milk at every feeding: he wants more in the morning, less in the afternoon. Therefore, the amount of milk the mother produces cannot be judged by weighing the child before and after one nursing alone; this control-weighing should be performed before and after all the feedings of a whole day. The average daily amount of milk needed by babies of different ages is as follows: at two weeks 500 ml, at one month 600 ml, at one and a half months 700 ml, at two months 800 ml, at three months 900 ml, and after five months one litre. More than one litre of milk, particularly cow's milk, should never be given to any child, whatever his age. The amount of milk imbibed is at first one-fifth to one-sixth of the child's total weight; in the second quarter of the year it is one-seventh of his weight. The daily amount of calories per kg of body weight is 120 in the first quarter of the first year, 110 in the

second quarter, and 100 in the third and fourth quarters. A litre of human milk contains approximately 700 calories. Some children take as much as 800 ml of milk in their second month of life.

The baby should be given only one breast at each feeding. No milk should be expressed from the breast before nursing the child; this should only be done after nursing. If the baby is weak, the milk should be expressed manually into a clean cup (rinsed with boiling water) and given to the baby in a spoon. If the mother has a small supply of milk she can give the baby both breasts at each nursing; all the milk left in the first breast should be withdrawn before the child is given the second. Then she empties the second breast too, and the milk thus expressed from both breasts is given to the child in a spoon. If the mammary gland is not emptied completely after each nursing the amount of milk will continue to decrease.

Mothers who do not have enough milk to nurse their babies can receive milk at the special milk banks set up at infant health centres, or they may obtain it from other nursing mothers; in the latter case the milk should be brought to a boil before it is given to the baby.

Sometimes children's hospitals use milk brought from home by the child's mother or by another woman. This milk must be tested every time, in order to safeguard the child against falsified milk—breast milk diluted with cow's milk.

The test is as follows: before the milk is boiled (1) its specific gravity is determined (to find out whether any water has been added), and (2) two drops of a neutral red solution (1 : 1,000) in an 0.85 per cent solution of table salt are added to 5 ml of the milk being tested. If it is pure human milk it colours orange, while if cow's milk has been added it becomes violet.

How is nursing to be managed in multiple deliveries—twins or triplets? The mother often has enough milk for both twins; she gives each child one breast at each feeding. Such a mother must drink three litres of liquid a day. However, if there is not enough milk for both babies, then the weaker one is given the breast alone, while the stronger twin gets supplemental food—formula or breast milk from another woman. Triplets usually get a mixed breast-formula diet: two babies get the breast at each feeding, while the third is given a bottle of formula or human milk, a strict rotation of bottle and breast being adhered to.

Mother's milk is full-value nutrition for the first six months of the baby's life. Breast-feeding without the addition of cow's milk

during this period is very important for the prevention of diarrhea and for lowering the child mortality rate. When the mother produces enough milk for her baby the early addition of formula and solids—at three or four months or even earlier—is absolutely unreasonable. If there is not enough milk additional food given during the first five-six months should be restricted to the usual milk formulas. After three months the baby should receive fruit or vegetable juices (black currants, tangerine, orange, tomato, carrots, black cherries); the fruits and vegetables must first be washed in hot boiled water. Up to six months babies are given a teaspoonful of juice three times a day, and a tablespoonful three times a day in the second half of the first year. The juices are sweetened to taste with sugar syrup. If the baby has a loose bowel movement all juices should be stopped. They must be introduced very carefully, so as not to upset the baby's digestion. After five months the baby may be given fruit kissel (a thin potato starch pudding made with sweetened fruit or berry juice) before the breast, one teaspoonful to six tablespoonfuls twice a day.

Adding Solids

At six months one breast-feeding, the 2 p.m. one, is replaced by a cereal (wheat farina); on the first day the child is given $\frac{1}{4}$ cup of cereal and the breast, the second day he gets $\frac{1}{2}$ cup of cereal and the breast, the third day $\frac{3}{4}$ cup of cereal and the breast, and on the fourth day a full cup of cereal and no breast. Thin cereal is gradually made thicker, and by seven months the baby is getting a tablespoonful of dry cereal to a glass of cooked cereal instead of the initial teaspoonful.

At seven months vegetable purée prepared with various vegetables, milk, butter, and sugar are added to the baby's diet, beginning with one teaspoonful and ending with three tablespoonfuls a day. By eight months one breast-feeding is replaced by a meal of cow's milk with white bread soaked in it, and also strained buckwheat or oatmeal instead of farina.

By 8-9 months the baby may be given half a cup (100 g) of meat or vegetable broth with strained vegetables and bread. The doctor may prescribe broth earlier in certain cases. By nine months a second breast-feeding is replaced by cow's milk or a roasted grain beverage with milk.

After eleven months the baby is given meat, at first as a meat purée (cooked meat twice forced through the meat grinder and then rubbed through a coarse sieve), later meat-balls in soup, and

after one year ground meat patties (hamburgers). Liver may be used instead of meat.

In the second half of the first year of life cottage cheese is recommended for babies after six months, beginning with 20 and ending with 50 g. Besides, nonallergic children may at this age be given eggs (one-half of a raw yolk beaten light, or hard-boiled, rubbed through a sieve and added to cereal or broth). Cereal with egg white may be recommended in cases where yolk is contraindicated, i.e., after a liver disease. The cereal is prepared in the following way: half an egg white (later a whole white) is mixed into a cooked and cooled cereal; when the white has blended in well (no shine on the surface) the cereal is again brought to a boil, and cooked for several minutes; while it is boiling the cereal is beaten with a spoon. Thus the white is given in a finely dispersed state and is well tolerated by babies. The best cereal to make with egg white is finely ground or pounded buckwheat.

The baby is weaned from the breast at the age of one year, but never in the summer, or during an illness, to avoid diarrhea. If the baby is weak it is desirable to continue giving him one or two breast-feedings after he is a year old, in addition to his regular solid meals. Breast-feeding in the second year of life is not harmful if the child receives full-value food—cereals, vegetables, fruit juices, etc. If the baby gets only the breast up to one year this may bring on anemia (due to the insufficient amount of iron in human milk).

Some institutions for very young children engage special wet-nurses who live in the same house with their children and produce an excess of up to a litre of milk daily for the sick babies. Before being engaged such a wet-nurse is examined for tuberculosis and syphilis. Her milk is tested every time and given without boiling. The age of the wet-nurse's child is of no account.

Mixed Feeding

Before supplemental solids are prescribed an attempt should be made to increase the amount of milk produced by the mother. To attain this the mother must always express any milk that remains in the breast after the child has nursed, must take enough liquid, sleep a sufficient number of hours, and get enough fresh air. If all these measures do not bring on the desired result the child is put on a mixed diet. It is best of all to give the baby human milk obtained at the infant health centre "milk banks", or from any nursing mother—but this milk *must* be boiled before it is given to

the child. Only in cases when it is impossible to get human milk should a formula be given (a mixture of cow's milk, water and sugar suitable to the baby's age). In no case should cereals be given; they are allowed only after 5-6 months. The formula is given as a supplement to every breast-feeding. The doctor prescribes the amount of formula to be given. To determine the necessary amount the baby is weighed before and after each of several test nursings in order to find out how much he can withdraw from his mother's breast.

The amount of milk necessary for one feeding is determined by the age and weight of the baby. Test weighing is performed two or three times. In certain cases, particularly when the mother has to work, one to three breast-feedings are replaced by bottle-feedings if the mother does not have enough milk.

In prescribing mixed feeding it should be remembered that the baby often takes the bottle much more readily than the breast, as it is easier to suck, especially if there is a large hole in the nipple. Therefore, the hole must be a small one—perforated with a red-hot needle—to make it just as difficult to suck the bottle as the breast, otherwise the baby may refuse to take the breast, the milk supply will accordingly decrease, and the baby will have to be gradually weaned to the bottle; this, in the absence of a source of good cow's milk, may lead to a severe illness. If the mother has enough milk for one or two nursings she should put the baby to the breast every time before she gives the bottle, as even a small amount of mother's milk is important for the proper development of the child.

Bottle-Feeding

Human milk and the milk of animals differ greatly in composition. Besides, the milk of animals becomes contaminated with various impurities during and after milking, and must therefore be boiled; boiling destroys a number of valuable constituents (vitamins, enzymes). No good results were reported when babies were fed on raw cow's milk. Alien protein, excessive amounts of casein and mineral salts, a different ratio of enzymes and vitamins, the opportunity for contamination in milking, changes caused by boiling—these are evidently the causes of the bad results of artificial feeding in 35 per cent of children. Giving the baby whole cow's milk in the first half-year of life is also not good for most children. And yet, of all animal milks the most suitable is cow's milk. In areas where goat's milk is widely used (in the South) babies may also be bottle-fed on goat's milk, only the goat's feed must be

balanced properly. The affirmation of many foreign authors that goat's milk allegedly causes anemia in children was not confirmed by Soviet investigators.

Anemia develops only when the child gets practically nothing else but goat's milk at an age when he should be receiving variegated foods. A similar condition might ensue if the child were fed only on cow's milk, or even only on breast milk at a time when he should already be getting solids.

Bottle-fed babies should be given only certified milk, conforming to the following requirements: the milk must come from a farm supervised by a veterinary, a sanitary doctor and a pediatrician: after the cow has been milked the milk is strained, cooled, pasteurised and sent off during the night in special refrigerator-cars by rail or motor transport. As little time as possible should pass between the milking and the delivery of the milk to the dairy kitchen of the infant health centre. In the dairy kitchen the milk is immediately strained, its acidity, fat content and specific gravity are assayed, and it is processed without delay. No more than 50,000 bacteria per ml are allowable in children's milk; its acidity should not exceed 20° (after Turner) (titration against N/10 alkali), and it must not curdle when a double amount of 68° alcohol is added to it; milk and milk mixtures should be bottled, the bottles stopped with cotton wool or porcelain plugs and put into a steriliser or kettle of cold water; the water is brought to a boil and the boiling continued for five minutes. The bottles are then cooled in cold water and stored on ice, or in the refrigerator. It is recommended to dispense the milk as soon as possible, and at home the mothers should keep it in the refrigerator, on ice, or in cold water, changing the water every hour. The milk must be cooled because boiling kills only bacteria, not their spores; if the milk remains warm, the spores again develop bacteria that decompose the milk components.

An infant deprived of mother's milk from the moment of birth is given a formula after six to twelve hours; this formula consists of cow's milk diluted by half with rice, barley, or oatmeal water, and 5 per cent sugar. The number of feedings and the daily quantity of formula is the same as in breast-feeding. When the baby is one month old it is given two-thirds milk and one-third cereal water.

As babies that are fed on cow's milk often develop rickets, they should be given fruit juices at two months; and, as a prophylactic measure, fish-liver oil (cod-liver oil) (beginning with 5-10 drops a day and bringing it up to two teaspoonfuls a day by adding

1-2 drops daily). Fish-liver oil is not given when the child has diarrhea, or in the hot season. Solids are added to the diet of bottle-fed babies in the same manner as to that of breast-fed babies.

The above simple formulas of cow's milk and cereal water may be prepared under almost any conditions and they have proved quite satisfactory in practice. Therefore, there is no need at the very beginning for the complex formulas that are now being prescribed less and less frequently.

If the baby does not thrive properly on the usual formula, it is given one or two bottles of diluted kefir (milk fermented by special kefir yeast), or simple formulas acidified with lactic acid, or curds (cottage cheese) rubbed through a sieve and added to rice water (one teaspoonful to one tablespoonful a day), or, finally, one to two teaspoonfuls of plasmon (a protein preparation) is added every day to the usual formula for a month or two. Plasmon is blended into two tablespoonfuls of formula, brought to a boil and then added to the bottle of formula.

Sometimes babies begin developing normally when they are given 1-2 bottles of a special fat formula instead of the ordinary one.

At first these formulas should be given with caution—no more than 50 ml a day. Fat formulas should be avoided in the summer, in hot weather, as they are poorly tolerated at this time and may easily lead to serious disorders in the child's digestion. When the baby has a tendency to looseness his physician sometimes prescribes protein milk for several weeks.

Tables 7 and 8 are approximate schedules for feeding infants. Table 9 gives the composition and caloric values of various milk formulas.

However, it must be stressed that these schedules are not strictly imperative for every child; practice has shown that individual traits call for some adjustment and variation. The proposed amounts of food can, of course, be highered or lowered.

Besides the solid foods in Tables 7 and 8, it is recommended to give children over eight months old meat or vegetable broth, no more than 100 ml. Broth is given either before or with vegetable purée and finely ground meat, depending on the child's individual taste.

Mixtures Mostly Used for Bottle-Feeding

1. *Cereal water* is prepared with rice, barley, pearl barley and oats. Forty g of the dry cereal and a pinch of salt are boiled in one litre of water for an hour; water is added all the time to make up for evaporation. The liquid is then strained off and water added to it to make up one litre. Whole wheat flour is used in the same way, but it is cooked for only 15 minutes. The amount of flour is prescribed by the doctor. When ready, the cereal water is sterilised in the usual manner in bottles (in the steriliser) for five minutes. Sometimes concentrated 10 per cent rice water is prescribed, the rice grains are strained, rubbed through a sieve several times and then added to the rice water.

2. *Sugar syrup*. One kg of granulated (or lump) sugar is added to 800 ml of hot water, under constant stirring. The mixture is brought to a boil and boiled until the sugar dissolves completely. Hot boiled water is then added to the syrup to make up one litre.

3. *Milk formulas with cereal water*. (B-rice, C-rice, and others). The freshly prepared cereal water is mixed with an equal part or two-thirds of cold raw milk; to this 5 per cent of concentrated sugar syrup is added, then the mixture is poured into bottles, sterilised in the usual manner, and cooled.

4. *Fat formulas*. Fat mixtures contain milk, sugar, and specially processed fat (fresh butter 1-3 per cent, concentrated cream 30 per cent). All the components are blended thoroughly, then sterilised in the usual manner and cooled.

a) *Fat-flour mixture No. 1*. Forty-six g of drawn butter is boiled in a pan over a slow fire until all the aliphatic acids volatilise (the smell of butter disappears); 46 g of wheat flour is browned on a frying pan, and then added slowly to the butter, stirring constantly, until a paste-like mass has been formed. To this mixture 665 ml of hot water with 33 g of sugar dissolved in it is gradually added, stirring briskly, until a homogeneous brownish mass is obtained; this mass is then strained, brought to a boil, and 335 ml of scalded milk is added to it; the mixture is then poured into sterilised bottles (not boiled any more) and cooled.

Mixture No. 2 contains 400 ml of milk, 600 ml of water, 30 g of sugar and 42 g of both butter and flour.

b) *Fat-flour milk formula* is made with acidified butter-milk. First the acidified buttermilk must be prepared (explanation lower). After the curds have set the whey is strained off through a sieve; the curds are stored in a sterilised container, the whey is mixed with sugar and gradually added, instead of milk, to the fat-flour mixture prepared as above. Flour and butter constitute 4 per cent and sugar 3 per cent of the mixture. Next the curds are rubbed through a sieve and added to the mixture, which is then poured into sterilised bottles. This mixture does not have to be sterilised.

5. *Acidified formulas*. a) *acidified buttermilk* is the product of clabbered milk from which butter has been churned, or of warm boiled skim or centrifuged milk fermented with a mixed culture of lactic acid bacteria, or with one tablespoonful of the previous day's buttermilk to one litre of milk; the milk is then placed in a thermostat at 32°C for 6-12 hours, or in a warm place. The buttermilk should not be too sour, as children do not take it readily; its acidity must not exceed 70° on titration against a N/10 solution of sodium hydroxide (after Turner), and 12° on titration against a N/4 solution of sodium hydroxide (after Soxhlet). The resultant buttermilk is warmed to 50°C; 5 per

cent sugar and 1-3 per cent flour are added to it, and sometimes 1-1.5 per cent fat; after this the buttermilk (stirring it constantly to avoid curdling) is poured into sterilised bottles, cooled, and stored in a cold place.

b) *Protein milk*. First method of preparation: to one litre of fresh milk a pinch of rennet powder is added and the milk is set in a pan for 10-15 minutes on a slow fire until the first curds appear; it is then strained, and the curds are put into a clean linen bag and hung up for half an hour to let all the whey drop off. If clabbered milk is used, one litre of the milk is placed over the fire and after the milk curdles it is treated as above. The curds are rubbed through a sieve several times, and 0.5l of boiled water is gradually added to it. The curds are then mixed with 0.5l of acidified buttermilk and the mixture is brought to a boil under constant beating and stirring. In preparing this milk experienced nurses make the curds so soft and fine that they pass through the nipple easily. In the absence of rennet powder whole boiled milk fermented with either lactic acid or kefir bacteria, or the previous day's buttermilk may be used. The curds are in this case also very flocculent. The protein milk is then poured into sterilised bottles and stored in a cold place. Three layers are formed in the bottle while it stands: the top layer is fat, the middle is greenish whey, and the curds are at the bottom.

c) *Protein milk*. Second method of preparation: one litre of milk is scalded and gradually cooled to 30°C. The milk curdles upon addition of rennet powder (0.05) blended into 30 to 50 ml of boiled milk. The mixture is kept at room temperature for half an hour, then placed over a low fire and heated to 40°C, till soft flocculent curds appear, then it is left to stand for half an hour; after that one litre of boiled water is added to it and the entire mixture is strained, the curds are rubbed through a sieve with a wooden masher. The liquid stands for two hours, then one litre is poured off without disturbing the curds that have settled at the bottom. The thus obtained protein milk is poured into sterilised nursing bottles, put in a pan of hot water (80°C), brought to a boil and boiled for five minutes; the bottles are taken out and vigorously shaken to break up the curds.

d) *Kefir*. Kefir is made with kefir grains (they consist of yeast and lactic acid bacteria). To make the leaven one tablespoonful of the kefir grains is put into a bottle of warm (boiled) skimmed milk (250 ml) and left to stand for six hours; the milk is then strained through a small sieve (that has first been scalded with boiling water); the grains are washed off with water and put into another bottle of fresh milk; this process is repeated 3-4 times; the last portion of milk is used for preparing the leaven proper: warm boiled milk is poured into nursing bottles, to each bottle 1-2 tablespoonfuls of the fermented milk is added, and the bottles are kept in a warm place for 24 to 48 hours. The leaven is now ready. For making kefir one teaspoonful of leaven is added to a nursing bottle of warm boiled whole milk. The bottles are placed in a warm place for 24 hours, and then in a cold place for another 24 hours. The kefir is ready in 48 hours (there are special kefir tablets that may be used instead of kefir leaven). Children are usually given 48-hour kefir, but in cases of constipation 24-hour kefir is used. Before giving the kefir to the child 5 per cent of sugar is added, and sometimes 5-10 per cent of cream (after Koltypin).

Two to five months babies are given kefir diluted with cereal water by half (B-kefir) or by one-third (C-kefir). For this purpose ordinary sterilised B-rice or C-rice with no sugar in it is fermented as described above, and before it is given to the child 5 per cent of sugar syrup is added.

Kefir is a very valuable nutrient, containing vitamin A; there are about 550 calories in one litre of kefir.

e) *Cottage cheese (pressed curds)*. Curds for cottage cheese are made from scalded milk in the same manner as described above for protein milk (with rennet, or from sour milk fermented with lactic acid bacteria). When the milk has curdled the curds are strained (the sieve is first scalded with boiling water), then rubbed through the sieve and placed in small sterilised jars, sealed and stored in a cold place. Cottage cheese spoils easily and should therefore be prepared fresh every day. Cottage (pot) cheese bought at the market is not suitable for infant-feeding.

f) *Acidified mixtures*. A 10 per cent solution of lactic acid is slowly added (drop by drop) to each nursing bottle of ordinary milk formula (8 ml^3 per 200 ml^3 of formula; after Luntz).

g) *Acidophilus milk*. Whole milk is poured into nursing bottles and sterilised in the usual manner for five minutes, and then inoculated with a pure (culture of *B. acidophilus* and placed for 5-6 hours in a thermostat at 40°C .

Preparation of Solid Foods for Babies

1. *Flour paste*. Thirty g of potato starch, wheaten flour or oatmeal are blended into 100 ml of cold water; the blend is stirred into one litre of hot milk, then 50 ml of sugar syrup is added and the whole mixture is boiled until it thickens.

2. *Fine wheat cereal (farina)*. Into a pan containing one litre of hot water 50 to 100 g of farina is added and the mixture is boiled down to one-third or one-fourth of the initial volume; then 650-750 ml of raw whole milk, 50 ml of sugar syrup, and a pinch of salt are added to the cereal and it is again boiled (practically this means one to three teaspoonfuls of dry farina to a glass of water). Buckwheat, oatmeal and rice are rubbed through a sieve after they have been boiled in water, and only then is the milk added; sometimes 25 g of fresh butter is put into the ready, half-cooled cereal. The prepared cereal is stored in sterilised nursing bottles.

3. *Fruit juice starch pudding (kissel)*. Kissel is a thin starch pudding made with sweetened fruit or berry juices. 100-150 g of fresh berries or apples are washed several times with hot water; the water is poured off and the berries are mashed with a wooden spoon or in a fruit press or juicer. The juice is poured into a sterilised vessel. Apples are first grated and then the juice is pressed out of them. The husks are boiled in a litre of water, and strained; 50-100 g of sugar and 25 g of potato starch are added to the liquid thus obtained (the starch should be stirred in 100 ml of cold water first) and the mixture is boiled until it thickens; when the pudding has cooled the juice that was first pressed out of the fresh fruit is added to it (to avoid decomposition of vitamin C). Kissel can also be prepared with dried berries (red and black cherries, black currants). Three tablespoonfuls of dried berries are cooked in one litre of water (the process is the same as above); when the pudding is ready any kind of fruit juice may be added. The kissel is poured into nursing bottles and stored in a cold place. Kissel may also be made with beet and carrot juice.

4. *Vegetable purée*. The vegetables (potatoes, carrots, beets, turnips, cabbage) are washed very thoroughly in water directly before cooking, and then placed in a steam-cooker (a pot containing a special basket or perforated stand) over a small amount of water; vegetables cooked over steam retain their valuable components. When soft the vegetables are peeled and rubbed

through a sieve, and milk and butter (according to the doctor's prescription) are added to them; the purée is warmed up before serving.

5. *Fruit and vegetable juice*. The raw juices of fruits, berries and vegetables have a high vitamin content. The fruits and/or vegetables are washed in hot water and mashed with a spoon or in a vegetable press or juicer; the juice obtained is strained through a piece of gauze or a fine strainer and poured into sterilised nursing bottles. Carrots and apples are first grated, then the juice is pressed out of them. Sugar syrup is added to taste.

6. *Stewed fruit (compote)*. Fresh grated, or dry, fruit may be used for stewing; when the fruit is cooked fruit juice is poured into it (as for kissel, only without starch).

7. *Meat broth (bouillon)*. 80-100 g of meat (leg or shoulder) and bones are scraped of fat and tendons and cut to pieces (the bones broken into small bits), placed in 1½ glasses of cold water over a moderate fire; the pot is then covered and slowly brought to a boil. The lid is then taken off and the pot moved to the edge of the stove (to simmer); when the froth rises in a compact mass it should be removed carefully, so that none of it settles at the bottom or remains floating. Three to five g of onions, 5-10 g of leeks, and 10 g each of parsley, carrots and turnips are cut into small pieces and put into the clear broth; when it comes to a boil the lid is again put on and the pot is left to simmer on the edge of the stove for 2-2½ hours. When ready, the broth must always be strained carefully and deprived of fat (after Kiselyova).

8. *Vegetable broth*. 70 g of yellow vegetables (carrots, turnips), 50 g of potatoes, 10-12 g of legumes (peas, beans) are cleaned, cut into large pieces (dry peas are ground first), placed in one litre of water and cooked for three hours in a closed pot, until only about 250-300 g of liquid is left. The broth is strained, the vegetables are not used. Salt is added to taste and the broth is again brought to a boil. Before serving, some fresh butter is put in (after Kiselyova).

9. *Patties*: a) Meat or fish fillet is put through a meat-grinder, and white bread added (half the amount of the meat); then balls are formed, flattened out and fried in butter, with a little broth added (soft steamed patties).

b) Patties made of sieved cooked vegetables are dipped in flour and fried in butter.

c) Patties made of thick cooked sieved cereal are also dipped in flour and fried in fresh butter.

Note: Fish-liver oil may be given to babies at three months, starting with 5-10 drops, adding 1-2 drops every day and bringing the dose to one teaspoonful. Fruit juice is introduced by first giving 5-10 drops in water, and then gradually increasing the amount until the baby is getting 10-20 g per meal.

Powdered Food Mixtures Used in Infant Feeding

At present powdered milk mixtures are commercially prepared for feeding babies; their ingredients are dried whole milk and various admixtures of dry cereals, vegetables, starch.

There also are on sale nutritive mixtures made of vegetable, fruit and other food powders, with or without dried milk, and also homogenised preserves.

Such mixtures, as proved by medical observations in various lands, are absolutely full-value indispensable products in baby-feeding, particularly in places where it is difficult to get good natural cow's milk.

Table 7

Recipes and Chemical Composition of Powdered Mixtures and Preserves for Babies

	Food product	Ingredients	%	Chemical composition			Calories per 100 g. of product
				Pro-teins	Fats	Carbo-hy-drates	
Milk formulas	Dry decoctions	Rice water	100.0	6.1	0.5	87.9	390.4
		Oatmeal water	100.0	14.8	7.1	70.0	413.5
		Buckwheat water	100.0	6.2	1.3	70.9	327.7
	B-rice	Whole dried milk	45.0				
		Sugar	35.0				
		Rice water	20.0				
		Total:	100.0	10.7	9.9	69.9	423.0
	C-rice	Whole dried milk	55.0				
		Sugar	33.0				
		Rice water	12.0				
		Total:	100.0	12.4	12.2	61.2	431.0
	B-oatmeal	Whole dried milk	45.0				
		Sugar	35.0				
		Oatmeal water	20.0				
		Total:	100.0	12.3	11.3	66.5	427.8
	C-oatmeal	Whole dried milk	55.0				
		Sugar	33.0				
		Oatmeal water	12.0				
		Total:	100.0	13.4	12.9	63.0	433.7
	B-buckwheat	Whole dried milk	45.0				
		Sugar	35.0				
		Buckwheat water	20.0				
		Total:	100.0	7.9	8.7	71.6	380.7
	C-buckwheat	Whole dried milk	55.0				
		Sugar	33.0				
		Buckwheat water	12.0				
		Total:	100.0	12.0	12.5	63.0	407.0
Farina cereals	Wheat farina 10%, on whole milk	Whole dried milk	46.0				
		Sugar	18.0				
		Farina	36.0				
		Total:	100.0	12.6	10.4	63.0	392.1

(Continued)

	Food product	Ingredients	%	Chemical composition			Calories per 100 g of product
				Pro-teins	Fats	Carbo-hydrates	
Farina cereals	Wheat farina 5%, on diluted milk	Whole dried milk	38.0				
		Sugar	31.0				
		Farina	31.0				
		Total:	100.0	10.6	8.6	68.3	387.8
	Milk starch pud- ding	Whole dried milk	60.0				
		Sugar	25.6				
		Starch	15.0				
		Total:	100.0	13.1	13.4	59.6	410.4

Table 8

Chemical Composition and Vitamin C Content in Nutritive Vegetable and Fruit Mixtures

Food product	Ingredients	%%	Chemical composition			Calor. per 100 g of product	Vitamin C content in mg %
			Pro-teins	Fats	Carbo-hydrates		
Rice-and-milk mix- ture with carrots	Dry powdered whole milk	47.0					
	Sugar	32.0					
	Rice flour	10.0					
	Dry powdered car- rots	7.0					
	Powdered dogrose hips concentrate	3.0					
	Salt	1.0					
	Total:	100.0	17.8	12.9	47.9	392.0	109.5
Vitaminised vege- table-flour mix- ture	Sugar	38.0					
	Rice flour	20.0					
	Oat flour	9.0					
	Buckwheat flour	9.0					
	Powdered dry car- rots	8.0					
	Powdered spinach	5.0					
	Powdered apples	5.0					
	Powdered dogrose hips concentrate	5.0					
	Salt	1.0					
	Total:	100.0	8.6	1.6	67.3	323.0	167.4

(Continued)

Food product	Ingredients	%%	Chemical composition			Calor. per 100 g of product	Vitamin C content in mg %
			Pro-teins	Fats	Carbo-hydrates		
Nutritive mixture of wheat flour and vegetables	Wheat flour, finely ground	86.0					
	Powdered carrots	4.0					
	Powdered spinach	5.0					
	Powdered dogrose hips concentrate	4.0					
	Salt	1.0					
	Total:	100.0	19.5	1.6	67.7	373.0	135.5
Nutritive mixture of rice flour and apples	Rice flour	90.0					
	Powdered apples	5.0					
	Powdered dogrose hips concentrate	4.0					
	Salt	1.0					
	Total:	100.0	10.5	1.4	75.2	366.0	139.4
Nutritive mixture of buckwheat flour and vegetables	Buckwheat flour	86.0					
	Powdered carrots	4.0					
	Powdered spinach	5.0					
	Powdered dogrose hips concentrate	4.0					
	Salt	1.0					
	Total:	100.0	19.9	6.3	65.1	407.0	122.7
Nutritive mixture of oat flour and vegetables	Oat flour	86.0					
	Powdered carrots	4.0					
	Powdered spinach	5.0					
	Powdered dogrose hips concentrate	4.0					
	Salt	1.0					
	Total:	100.0	19.9	6.3	65.1	407.0	122.7

Table 9

Schedule of Breast-Feeding and Addition of Solids

Age	Number of feedings	Hours	Food	Amount of breast milk and other foods in g	Total daily amount (in g)	
					Breast milk	Other foods
Up to 1 month	7	6	Breast	70-100	500 to 700	
		9	ditto	70-100		
		12	ditto	70-100		
		15	ditto	70-100		
		18	ditto	70-100		
		21	ditto	70-100		
		24	ditto	70-100		
1 to 3 months	7	6	Breast	100-120	700 to 840	
		9	ditto	100-120		
		12	ditto	100-120		
		15	ditto	100-120		
		18	ditto	100-120		
		21	ditto	100-120		
		24	ditto	100-120		
3 to 5 months	6	6	Breast	140-150	840 to 900	Fruit juice 20-40 ml Fish-liver oil 2 tsp.
		9.30	Breast	140-150		
			Fruit juice	10-20		
		13	Breast	140-150		
			Fish-liver oil	1 tsp.		
		16.30	Breast	140-150		
			Fruit juice	10-20		
		20	Breast	140-150		
			Fish-liver oil	1 tsp.		
		23.30	Breast	140-150		
5 to 6 months	5	6	Breast	180-200	800	Fine wheat cereal 150 g Kissel 50-100 g Fruit juice 50 g Fish-liver oil 2 tsp.
		10	5% wheat farina	150		
			Kissel	50-100		
			Fruit juice	30		
		14	Breast	180-200		
			Fish-liver oil	1 tsp.		
		18	Breast	180-200		
			Fish-liver oil	1 tsp.		
		22	Breast	180-200		
			Fruit juice	20		

(Continued)

Age	Number of feedings	Hours	Food	Amount of breast milk and other foods in g	Total daily amount (in g)	
					Breast milk	Other foods
6 to 7 months	5	6	Breast	200	600 to 800	Cereal, 150 g Kissel 100-150 g Fish-liver oil 2 tsp. Fruit juice 50 g Whole milk or kefir 150 g
		10	10% wheat farina	150		
			Kissel	50-100		
			Fruit juice	30		
		14	Puréed vegetables	50		
			Breast, whole milk or kefir	150		
			Fish-liver oil	1 tsp.		
		18	Kissel	50		
			Breast	200		
			Fish-liver oil	1 tsp.		
		22	Breast	200		
			Fruit juice	20		
7 to 8 months	5	6	Breast	200	400 to 600	Cereal, 200 g Kissel 150-200 g Whole milk or kefir 200 g Potato purée 100 g $\frac{1}{2}$ egg yolk Fruit juice 50 g Fish-liver oil 2 tsp.
		10	Wheat farina	200		
			Kissel	50-100		
			Fish-liver oil	1 tsp.		
		14	Puréed potatoes	100		
			Egg yolk	$\frac{1}{2}$ yolk		
			Kissel	100		
			Fruit juice	30		
		18	Breast, whole milk, or kefir	200		
			Fish-liver oil	1 tsp.		
		22	Breast	200		
			Fruit juice	20		
8 to 11 months	5	6	Breast	200	400	Cereal, 200 g Fruit juice 50 g Fish-liver oil 2 tsp. Cottage cheese, 30-50 g
		10	Cereal	200		
			Scraped apple or fruit juice	20-40		
			Fish-liver oil	1 tsp.		

(Continued)

Age	Number of feedings	Hours	Food	Amount of breast milk and other foods in g	Total daily amount (in g)	
					Breast milk	Other foods
		14	Vegetable purée	150		Kissel 150 g
			Cottage cheese	30-50		Whole milk or
			Kissel	100		kefir 200 g
			Cracker or dry toast	10		.
		18	Whole milk or kefir	200		
			Fish-liver oil	1 tsp.		
			Cracker	10		
			Kissel	50		
		22	Breast	200		
			Fruit juice	30		
11 to 12 months	5	6	Breast, whole milk, or kefir	200	200	Whole milk or kefir 200-400g
			Fruit juice	40		Cereal 200 g
			Cracker or dry toast	10		Kissel 200 g
		10	Cereal	200		Fruit juice 70 g
			Kissel	100		Scraped apple or stewed fruit 150 g
		14	Fish-liver oil	1 tsp.		Ground meat 30 g
			Purée potatoes	150		Cottage cheese 50 g
			Ground meat	30		Potato and vegetable purée 250 g
			Fruit juice	30		Fish-liver oil 2 tsp.
			Stewed fruit or raw apple	100		
				50		
		18	Cottage cheese	50		
			Vegetable purée	100		
			Kissel	100		
			Fish-liver oil	1 tsp.		
		12	Whole milk or kefir	200		
			Cracker or dry toast	20		
			Bread	50		

Note: Babies over 8 or 9 months may be given other cereals than fine wheat (farina): buckwheat, oatmeal, rice, millet.

Schedule of Artificial Feeding and Addition of Solids

Age	Number of feedings	Hours	Food	Amount of milk and other food in g	Total daily amount (in g)	
					Milk	Other foods
Up to 1 month	7	6	B-rice	50-90	350 to 630	
		9	ditto	50-90		
		12	ditto	50-90		
		15	ditto	50-90		
		18	ditto	50-90		
		21	ditto	50-90		
		24	ditto	50-90		
1 to 3 months	7	6	C-rice	100-150	700 to 900	Fruit juice 10 g Fish-liver oil 1 tsp.
		9	ditto	100-150		
			Fruit juice	5		
		12	C-rice	100-150		
		15	ditto	100-150		
			Fish-liver oil	½ tsp.		
		18	C-rice	100-150		
			Fish-liver oil	½ tsp.		
		21	C-rice	100-150		
			Fruit juice	5		
		24	C-rice	100-150		
3 to 5 months	6	6	C-rice	150-180	900 to 1,080	Fruit juice 20 g Fish-liver oil 2 tsp.
		9.30	C-rice	150-180		
		13	C-kefir	150-180		
			Fish-liver oil	1 tsp.		
		16.30	C-kefir	150-180		
			Fruit juice	10		
		20	C-rice	150-180		
			Fish-liver oil	1 tsp.		
		23.30	C-rice	150-180		
5 to 6 months	5	6	C-rice or whole milk	180-200	900 to 1,000	Fine wheat cereal 150 g

(Continued)

Age	Number of feedings	Hours	Food	Amount of milk and other food in g	Total daily amount (in g)	
					Milk	Other foods
		10	Fruit juice 5% wheat farina Kissel	20 150 100		Fruit juice 50 g
		14	Fish-liver oil Kefir or whole milk	2 tsp. 180-200		Fish-liver oil 1 tsp.
		18	Fruit juice Kefir or whole milk	30 180-200		
		22	Fish-liver oil Whole milk	1 tsp. 130-200		
6 to 7 months	5	6	Whole milk	200	750	Cereal 150 g
		10	10% wheat farina Kissel	150 100		Kissel 100 g
			Fish-liver oil	1 tsp.		Vegetable pu- rée 50 g
		14	Vegetable pu- rée Kefir	50 150		Fish liver oil 2 tsp.
			Fish-liver oil	1 tsp.		Fruit juice 50 g
		18	Kefir	200		
			Fruit juice	20		
		22	Whole milk Fruit juice	200 20		
7 to 8 months	5	6	Whole milk	200	600	Cereal 150 g
			Cracker or dry toast	10		Kissel 100 g
		10	Cereal Kissel or stewed fruit	150 100		

(Continued)

Age	Number of feedings	Hours	Food	Amount of milk and other food in g	Total daily amount (in g)	
					Milk	Other foods
		14	Fish-liver oil	1 tsp.		
			Potato purée	150		Dry toast or cracker 20 g
			Cottage cheese	30-50		
			Fruit juice	30		Cottage cheese 30-50 g
		18	Kefir	200		Fruit juice 50 g
			Fish-liver oil	1 tsp.		Fish-liver oil 2 tsp.
			Cracker or dry toast	10		
		22	Whole milk	200		
			Fruit juice	20		
8 to 11 months	5	6	Whole milk	200	600	Cereal 200 g
			Cracker or dry toast	20		
		10	Cereal	200		Kissel 100 g or stewed fruit 100 g
			Kissel or stewed fruit	100		Cracker or dry toast 40 g
			Fish-liver oil	1 tsp.		
		14	Potato purée	200		
			Cottage cheese	50		
			Fruit juice	30		Fruit juice 50 g
		16	Kefir	200		
			Cracker or dry toast	20		Fish-liver oil 2 tsp.
		22	Fish-liver oil	1 tsp.		
			Whole milk	200		
			Fruit juice	20		
11 to 12 months	5	6	Whole milk	200	400	Cereal 200 g

(Continued)

Age	Number of feedings	Hours	Food	Amount of milk and other food in g	Total daily amount (in g)	
					Milk	Other foods
			Cracker or bread	20-30		
			Fruit juice	30		Kissel 200 g or stewed fruit
			Cereal	200		Potato purée 150 g
			Kissel or stewed fruit	100		Vegetable purée cooked in meat broth 100 g
			Fish-liver oil	1 tsp.		Ground meat 30 g
		14	Potato or vegetable purée	150		
			Ground meat	30		
			Kissel or stewed fruit	100		
		18	Fruit juice	20		
			Vegetable purée cooked in meat broth	100		½ egg yolk in purée or cereal
			Bread	30		Crackers 40 g
			Egg yolk	½ yolk		Bread 30 g
			Fish-liver oil	1 tsp.		Fruit juice 50 g
		22	Kefir	200		Fish-liver oil 2 tsp.
			Cracker or dry toast	20		



Table 11

Composition and Calories of Food Mixtures
(Cal. per litre)

Food Mixture	Proteins	Fats	Carbohy- drates	Calo- ries
		(in g)		
Human milk	15	40	70	700
Cow's milk	34	37	45	668
Whole milk + 3% sugar	34	37	75	790
Mixture No. 2: 1/2 part whole milk + 1/2 part sugar + 5% sugar	17	18	72	540
Mixture No. 3: 2/3 whole milk + 1/3 water + + 5% sugar	22	24	79	600
B-rice: 1/2 part whole milk + 1/2 part 4% cereal water	17	18	76	555
C-rice: 2/3 whole milk + 1/3 4% cereal wa- ter	22	24	79	660
Fat-flour mixture No. 1	11	58	48	945
Protein milk	30	25	20	437
Protein milk + 5% sugar	30	25	70	642
Protein milk + 5% sugar + 3% flour	30	25	100	745
Buttermilk	35	5	40	353
Buttermilk + 5% sugar + 3% flour	35	5	120	681
Kefir	30	30	35	550
Sour (clabbered) milk	30	27	30	500
Cottage cheese (1 kg)	138	6	26	705
Dubo mixture (milk + 17% sugar)	34	30	215	1,260
4% rice water	1.0	0.5	14	62
10% rice water			74	335
Fine wheat cereal (farina), 5% (750 ml milk + + 5% sugar)	31	31	113	866
Fine wheat cereal (10% + 5% sugar) . . .	36	32	113	895
Fruit kissel (125 g berries + 50 g sugar + 25 g flour)	0.5	0.3	72	293
Flour paste cooked with whole milk + 5% sugar	34	37	114	952
White bread (1 kg)	285	4	464	2,260
Sweetened apple purée (+5% sugar) . . .	3	1	150	650
Carrot purée	12	3	90	485
Tangerine juice	8	—	126	540
Tomato juice	13	2	40	238
Carrot juice	12	3	90	435

Feeding Children After One Year

Researchers have evolved standard norms showing the dietary requirements of children over one year of age in proteins, fats and carbohydrates.

In 1951 the U.S.S.R. Ministry of Public Health approved the norms of nutrition for children presented in Table 12. These norms were computed by the Nutrition Institute of the U.S.S.R. Academy of Medical Sciences.

Table 12

**Standard Physiological Dietary Requirements of Children
and Adolescents in Proteins, Fats, Carbohydrates and Calories
(g per day)**

	6 months to 1 year	1 to 3 years	3 to 7 years	7 to 11 years	11 to 15 years	15 to 18 years
Proteins	25	48	68	78	98	119
including animal proteins . . .	21	39	48	50	56	72
Fats	25	51	65	81	86	99
including animal fats	25	50	61	72	75	84
Carbohydrates	109	157	241	297	424	471
Calories	732	1,315	1,871	2,291	2,940	3,340
including calories from animal products	396	727	855	966	1,031	1,181

Mineral substance requirements: the child's food should include, daily, 1 g of calcium, 1.5 to 2 g of phosphorus, 0.15 to 0.2 g of iron, and 0.05 g of sodium chloride per kg of body weight.

Mineral substances occur in 1) foods of animal origin (milk, meat, eggs); 2) in grain products (cereals, bread); 3) in vegetables and greens.

Water requirements: adults and older children need a minimum of 40 to 50 ml of water per kg of weight, but babies require no less than 100-150 ml per kg of weight.

Soup is given as a liquid food in the following amounts, depending on the age of the child: from one to three years no more than 200 to 250 ml, from three to eight years 300 to 350 ml, and school children receive 350 to 500 ml of soup.

Children should be taught to drink water slowly, as drinking in large gulps stimulates cardiovascular activity too much.

Children over one year old are given four meals a day.

The daily food ration of healthy children (in home conditions) should be portioned out as follows: the morning meal (breakfast)

25-30 per cent, the mid-day meal (dinner or lunch) 40-50 per cent, the afternoon snack 10-15 per cent, and the evening meal (dinner or supper) 15-20 per cent of the daily ration.

School children who get hot meals at school should be given 15-20 per cent of their daily ration in the morning, 25 per cent at 11 or 12 a.m. at school, 40 per cent after school (dinner), and 20 per cent in the evening (supper). Tables containing the chemical components and calories of various foodstuffs are used for making up daily menus (Table 13).

Table 13

Chemical Composition and Caloric Values of Various Foods

Food	Proteins	Fats	Carbohy- drates	Calories
	(per 100 g)			
Milk	3.4	3.7	5.0	64
Brown (rye) bread . .	6.5	0.6	51.2	236
White (wheat) bread	5.5	0.3	56.0	251
Meat, raw	20.0	4.8	—	123
Fish	18.0	0.7-1.0	—	82-100
Butter	1.0	83.0	0.5	753
1 egg	5.6	5.4	0.2	75
Sugar	—	—	99.7	410
Cottage cheese	14.7	0.6	2.2	125
Kefir	3.7	3.2	3.6-0.4	66
Potatoes	2.0	0.1	22.5	100
Cabbage	1.8	0.2	4.8	30
Cucumbers	1.1	—	2.3	15
Apples	0.3	—	12.0	51
Grapes	0.5	—	16.0	69

Protein-containing food (meat, fish, legumes) should be given to the child in the morning and afternoon; the evening meal should be a light one, with no meat nor fish.

Young children are given 75 per cent of white and 25 per cent of brown bread, school children get 50 per cent of each.

Vitamins are also very important items. They are required not only for counteracting the development of various vitamin deficiencies, but also as protection against infectious diseases.

In this respect vitamin A is particularly important; it occurs in carrots, eggs, milk, fresh butter, and some other foods.

The child usually gets his vitamin C with raw fruits and vegetables and their juices.

The vitamin B group occurs in meat, kefir, cottage cheese, legumes and cereals; these vitamins are likewise very necessary,

as they are an important factor in the activity of the child's nervous system.

Vitamin D is necessary for proper bone formation and to prevent rickets; this vitamin is contained in fats, eggs, and other products of animal origin.

Nutrition of children from one to three years. During this period the child's milkteeth develop, he starts walking, becomes more active. Therefore, at this age children need coarser food. However, as the masticatory organs are not fully developed yet, the child's food should be prepared accordingly (chopped and strained).

The diet of a child from one to three years may be quite variegated, but milk still makes up a significant part of it—from 500 to 600 ml; milk may be given in the form of kefir, clabbered milk, curds. The child gets various cereals (wheat cereal [farina], rice, millet, buckwheat), baked puddings. He may be given both meat and vegetable soups cooked with cereals and vegetables. He is also given fish, meat no oftener than once a day (meat-balls in soup, or meat patties, meat purée), eggs (no more than one every other day), mashed potatoes. Vegetables should be finely chopped (salads, purées); vegetables must not be cooked too long as this destroys a significant part of the vitamin C. Fruits are given raw and stewed, in fruit-mousse and starch puddings (kissel). The child may have both white and brown bread of various sorts, bread and butter, bread and jam; roasted grain beverages may be given with milk. Of sweets children at this age are given sugar, jam, fruit preserves, honey, marmalade.

Children should be taught to be neat and tidy; they should learn to feed themselves with a spoon, put on their pinafores, wash their hands before meals.

Nutrition of children from three to seven years (preschool age). Within this period of life the teeth and mastication fully develop, so the child may be offered *hard foods*. The protein norm (approximately 3 to 4 g per kg of weight) should be adhered to strictly. The child is already able to feed himself, and may have his meals with the rest of the family. Care of the mouth and teeth is very important (the child should have his own tooth-brush and be able to use it). At this age children should be getting about 500 ml of milk and dairy products; more meat and eggs may be given; the food is not chopped or strained as carefully as before. Both rye and wheat bread is given, weak tea and roasted grain beverages, sugar, fruit preserves, honey, marmalade. An abundance of fruit and vegetables, legumes, cabbage, lettuce, spinach, cereals, fruit and berry starch puddings, stewed fruit are highly recommended.

Nutrition of children from seven to fifteen years of age (school age). The food at this age approaches that of adults. The school-child should have no less than 500 ml of milk and dairy products a day, and meat once or twice. He should be given full-value proteins in sufficient amounts, and his food should be of a mixed nature (meat, vegetables, fruit), no less than two hot meals a day.

Irritating substances—pepper, mustard—are forbidden for children of any age, as are wine and beer. Children's food should be fresh, well and tastily prepared, sufficiently variegated, served hot.

Public catering has become very popular in the Soviet Union. This includes food for babies in nurseries (*crèches*) additional solids dispensed for young children by *special dairy kitchens* and infant health centres, the meals at round-the-clock nurseries. In the summer children are fed at *camps*, and *summer colonies*. Besides this, there are special *dietary canteens* for children, meals served at special health-promoting playground centres where weaker children spend part of the day. Wide-scale development of public catering for children is a leading factor in promoting the health of the child population, and is very important in lowering the child morbidity and mortality rates. An important factor in nutrition is just how and where children's meals are served.

According to the teachings of Pavlov, proper digestion and assimilation of food is greatly facilitated by regularity in mealtimes, whereby the alimentary tract receives timely stimuli of definite intensity from a given food.

It was demonstrated by Pavlov and his associates that the entire environment and the aspect of the food stimulate the secretion of the most effective digestive juice, the so-called "priming" juice.

When nursing time approaches a breast-fed baby has only to see its mother's uncovered breast to secrete this juice.

A bottle of milk or red kissel evoke the secretion of the "priming" juice in babies who are on formula, or who are given additional food. The alimentary glands of babies can only function properly if the food the child gets conforms to the facility of these glands to digest it.

Breast milk, and later cow's milk, call for the least effort on the part of the digestive glands. Milk remains one of the staple foods in the subsequent life of the child.

It is important that the food of older children, both at home and in various child-care institutions, be variegated and taste well. Meals should be a quiet affair. The very look of the table on which the food is served is also important.

Radish, lettuce, greens and vegetables are suitable appetisers for children, as they are not too pungent. Appetisers greatly facili-

tate the secretion of the most active portion of gastric juice. The following dish should be served after not too long an interval, otherwise the child's attention is distracted by something else and he loses his appetite.

CHILDREN'S NUTRITION IN ILLNESS

The management of the feeding of sick children is a very responsible job. The general and basic stipulation is that the child gets a *sufficient amount of food of full nutritional value*. Nutrition is very important in the therapy of disease, and it should be given proper attention. The sick child has often to be patiently fed and encouraged to eat. At times nutrition must be introduced through a tube (in diseases of the brain, hysteria, and a number of nervous diseases), by nutritive enemas, or injected (5 per cent glucose) subcutaneously or intravenously. However, a number of diseases call for a temporary discontinuance of food, for its limitation, for special diets, when some foods are temporarily excluded (for instance, salt in nephritis). During convalescence such patients require particularly nutritious food and additional vitamins and fats.

Nutrition in Gastrointestinal Diseases

The chief treatment of gastrointestinal diseases is diet. A basic rule is *temporary* hunger (6-12-18 hours) in the early phases of the disease, putting the child on a water or tea diet. He is given only tea or water by spoonfuls, a 5 per cent glucose solution, physiological saline or Ringer solution (100 to 200 ml). These measures must be observed in simple or toxic dyspepsia in infancy, in dysentery at an early age if there is vomiting, in food poisonings accompanied by vomiting, in uremia, in incipient nephritis.

Severe conditions accompanied by vomiting necessitate the introduction of fluids into the system—Ringer solution or physiological salt solution (normal saline solution) in enemas or subcutaneously (200-300 ml, depending on age).

Later a more substantial diet is prescribed, depending on the type and severity of the disorder. It must be remembered that the diet of young babies suffering from *acute*, and particularly *toxic* dyspepsias must be rationed very cautiously. They are given breast milk expressed manually, starting with 5-10 ml every two hours, and adding 5 ml per feeding every day. Breast milk is the main food, lactic acid mixtures take second place, kefir is given still later.

The first phase of dysentery (first to fourth day) is accompanied by vomiting and acute intoxication; children, particularly infants, should be given very limited quantities of food and put on a water diet. Subsequently, even if loose bowel movements with mucus and blood persist, the child must not be starved; his food should contain a sufficient amount of nutritive substances, proteins, carbohydrates, vitamins, and be easily assimilated and digested.

Thus dangerous emaciation and grave complications are avoided (noma, nonprotein edemas). These rules are also true for diseases such as typhus, typhoid and paratyphoid, in which food of full dietary value is also an important therapeutic factor.

Nutrition in Heart Diseases and Rheumatism

Children afflicted by heart diseases or rheumatism need rich, vitamin-containing food. Monotonous starchy foods (bread, porridge, starch puddings) should not be given too abundantly, as such a diet intensifies the tendency to water retention in the tissues that usually accompanies these disorders. The child's diet should have a normal content of all ingredients and include fresh fruit and vegetables; fish-liver oil is prescribed.

Nutrition in Nervous Disorders

Children suffering from meningitides and meningo-encephalitis are frequently poor eaters and refuse food; in grave cases they cannot even swallow. Such children must be fed, patiently and very persistently, on rich, easily digested food. Temporary deglutitory disorders sometimes necessitate nourishment (particularly in the case of infants) through a tube. This feeding technique alleviates the course of such grave diseases. The same pertains to diphtherial paralyses accompanied by difficulty or inability to swallow.

Nutrition in Skin Diseases and Exudative Diathesis

It has been proved by practice that sharp modifications of diet (exclusion of proteins or fats, limitation of carbohydrates) are of no help in exudative diathesis. Overfeeding (particularly with milk) is harmful. The patient should be given variegated foods of sufficient dietary and vitamin value. Infants afflicted by exudative diathesis must not be deprived of breast milk—as is often practised—as it is highly necessary for them.

Nutrition in Acute Infections

In acute infections the patient needs a sufficient amount of food with high dietary values. However, this is prevented by the general *intoxication* observed at the onset of the disease, accompanied at times by loss of consciousness followed by *the development of a local process* in the throat (diphtheria, scarlet fever, pharyngeal conditions, chickenpox), or in the glands (mumps, tonsillitis). The same is observed in disorders of the central nervous system (meningitis, encephalitis, tetanus). In all these diseases the patient should be given easily assimilable semiliquid food of sufficient dietary value. The fluid balance is also very important; if the patient cannot drink enough water, it is introduced either through the rectum or subdermally. In the convalescent stage variegated food and fish-liver oil are prescribed. During the entire period of disease and recovery fruit and vegetable juices are recommended, fresh and cooked, and acidified drinks.

Nutrition in Chronic Illnesses

The diet of tuberculous patients depends on the form of the disease. Often tuberculous children lose their appetites and nutrition is much lowered; parents try to feed the child excessively, this calls forth a still greater aversion to food, and often causes vomiting. The patient should never be forced to eat, but he should be encouraged to take the necessary (not excessive) amount of wholesome, variegated, tasty food. The child's ration should include fruit, vegetables, and fish-liver oil. Fresh air improves the appetite, so the child should be out-of-doors as much as possible. A similar regimen should be observed in other chronic debilitating diseases (malaria, lymphogranulomatosis [Hodgkin's disease], bronchiectasis).

Diet in nephritis is based on a temporary exclusion of ingredients that are not excreted by the kidneys. In the acute period this includes water that should be restricted for a day or two, and salts that must be excluded temporarily or restricted in the acute period when the disease is accompanied by *edema*.

It must be remembered that salt cannot be excluded for prolonged periods. There is absolutely no need in *pyelitis* for the dietary restrictions prescribed in nephritis (as is at times practised). Temporary limitations in diet are necessary in diseases such as jaundice, certain endocrine disorders (tetany), and others described in the corresponding chapters of this book.

Public catering for sick children is practised in hospitals, clinics, sanatoriums, special health-promoting boarding-schools in the country (so-called "forest schools"), open-air schools, health-centre playgrounds. In these institutions the children are given either full (all-day) or part-time nutrition, depending on age and type of disorder. The correct management of nutrition is one of the basic factors of treatment.

PRINCIPLES OF CARE FOR HEALTHY NEWBORN INFANTS AND NURSLINGS

A basic requirement in the care of the newborn is scrupulous cleanliness and aseptic (sterile) conditions during delivery. The subsequent health of the baby depends to a large extent on the quality of the work of the obstetric staff. The mucous membranes and the skin of the newborn are easily infected by pyogenic microbes that facilitate the development of septic diseases. The appearance in maternity hospitals of various types of toxico-septic diseases in the newborn are for the most part a sign of poor sanitary and hygienic conditions in this institution, of the negligence and oversight of its staff. The newborn child may be infected through the hands of the staff and through various objects by staphylococci, streptococci, *Escherichia coli* (*Bacillus coli*), pneumococci and other pathogenic microbes. The staff of maternity hospitals should always wear gauze masks, changing them daily; there always should be a sufficient amount of such sterilised masks in these hospitals.

As soon as the child is delivered, his eyes are treated after the Matveyev method. The midwife (or doctor) washes her hands and rubs them with alcohol, then instills one drop of a 2 per cent solution of silver nitrate into the conjunctival sac of each eye, pulling down the lower eyelid with the left hand, after which each eye is dried separately with a clean swab of absorbent cotton.

Five to ten minutes after birth, when strong pulsations of the umbilical vessels have ceased, the cord is tied, in two stages. First it is ligated with a narrow strip of sterile gauze at a distance of 10 cm from the umbilical ring, then a second ligature is placed 3-4 cm higher. The length of cord between the two ligatures is swabbed with alcohol and then cut with sterile scissors. The surface of the cut is painted with iodine, and a sterile gauze napkin is placed over the protruding end of the cord.

The newborn infant is then wrapped in a diaper and placed on the diapering table, where a second umbilical ligation is performed,

at a distance of 1.5 cm from the umbilical ring. The long end of the cord is swabbed with alcohol and cut off, the wound dressed with iodine and covered with two-three layers of gauze napkin. The umbilical stump, tied with gauze, is not bandaged, as was done formerly, but left free.

After this procedure the newborn is placed on a heated rubber pad (hot-water bottle), and the superfluous waxy deposit (vernix caseosa) in the creases of the neck and in the armpits is carefully removed with a cotton swab dipped in sterile mineral oil; the skin of the inguinal region is cleaned of mucus and blood.

The body wax should not be removed entirely, as it protects, to a certain extent, the skin against maceration. Later it is partly absorbed and partly deposited on the diapers.

After the baby's toilet has been completed, all its natural orifices are examined—the nostrils, ears, anus, the opening of the urethra—and it is ascertained that there are no fractures, abrasions nor scratches on the skin; the condition of the caput succedaneum (the molding hemorrhage on the head) is observed, the infant is examined for any abnormalities that may be present. After this the height (crown-heel length) and circumference of the head are measured and the baby is weighed; all these data are entered into the development record.

After all the above procedures the baby is dressed, and approximately two hours after delivery, while the mother is still in the delivery room, it is transferred to the nursery department of the hospital. A sterile oilcloth or plastic bracelet is placed on the baby's wrist; on it are written the name and surname of the mother, the sex of the child, date of birth and number. A card carrying the mother's number is hung on the baby's crib.

Further the newborn baby is cared for by special nurses supervised by a pediatrician (in the nursery).

The pediatric nurse looks after the baby, observes its condition, makes its daily toilet, washes its diaper area, and applies sterile mineral oil to the creases in its skin. The diaper area should be washed with warm water (at body temperature) after every bowel movement or urination. The genitalia of girls are washed from front to back in order to avoid the introduction of any infection to the urethra; the skin is blotted after washing (not wiped) with a dry diaper.

It is necessary to see that the bandage on the cord holds securely, and that there is no redness around the navel.

On the fifth day after birth the navel is examined in the doctor's presence, and another dry dressing is placed on it. The cord usually

dries up and falls off while the baby is still in the hospital, on the sixth-seventh day of life. When it has fallen off the little wound that remains is painted with a 2 per cent silver nitrate solution; and the navel is covered with dressing until the wound has closed completely. If any irritation is noticed on the baby's skin, measures are taken to heal it. Slight irritations are treated by washing the area with a weak solution of potassium permanganate and then applying sterile mineral or fish-liver oil; in more serious cases, when maceration of the skin has commenced, special ointments or salves are prescribed by the doctor.

It is not recommended to wrap the baby up in his diapers together with his rubber or oilcloth pad, as this easily leads to the maceration of the tender skin on the buttocks and groin, and this may cause a severe skin irritation.

The baby's face is washed every day with warm water, his eyes are wiped with a swab of cotton dipped in boiled water or a 2 per cent boric acid solution, from the outer corners of the eye to the nose. The ear is cleaned with a dry twist of cotton (don't use a match-stick!), and the nose with a twist of absorbent cotton dipped in mineral oil.

The child's mouth does not have to be cleaned, as the oral mucosa is easily injured and this, in its turn, may pave the way for monolial infection (thrush) (see p. 201). The nurse in the pediatric section of the maternity hospital takes the temperature of the newborn every day; in normal children delivered at term the temperature directly after birth is elevated, being 37.7 to 38.2° (in the rectum), but in a very few hours this temperature goes down, becoming normal at the end of the first 24 hours, 36.6 to 37°. The temperature may sometimes suddenly go up to 38-39° and even higher on the third or fourth day of life. This is the so-called *transitory or dehydration fever of the newborn*; it is probably due to an inadequate fluid intake. If the feeding schedule is managed properly, and the infant gets enough fluids orally (water, tea, glucose solution) this elevation of temperature has no significant effect on his general state, and improvement is rapid. Naturally, it should always be remembered that an elevated temperature in the newborn may be due to some incipient disease. Therefore, in order to determine the nature of such temperature elevations it is necessary to exclude the possibility of disease in every given case (sepsis, pneumonia, etc.).

During the entire subsequent period of infancy a temperature characteristic is established; it is distinguished by no noticeable vacillations (monothermy), the difference between the morning and

evening temperatures not exceeding 0.3° . However, it must be noted that overwarming may cause an elevation of temperature in babies, as their central nervous system does not yet effect normal heat regulation. The child may be overheated from being overdressed, particularly in the hot season, or by being kept in a stuffy room, or through excessive use of hot-water bottles. Overcooling may lead to a fall in the temperature lower than normal, causing the subdermal fat tissues to harden (sclerema).

In the maternity hospital the baby is not bathed, as baths are permitted only after the cord has withered and sloughed. The first



Fig. 12. Bathing an infant

bath is given at home, 24 hours after the cord has fallen off. The temperature of the water must not be lower than $36-37^{\circ}\text{C}$, while the temperature of the surrounding air should be no lower than $19-20^{\circ}\text{C}$. When the baby is immersed into his bath his head is supported on the left arm, his back being held (under the arm) by the left hand (Fig. 12). The body of the baby should be covered with water up to his shoulders; he is kept in the bath 5-10 minutes. Soap (specially for children—a mild white soap) is used no oftener than once a week, while on the rest of the days he is bathed in water alone. After the bath the baby is dried carefully in a warm soft towel or sheet that has been washed quite a few times; all the creases of the baby's skin are swabbed with sterile mineral or vegetable oil (sunflower-seed, cotton-seed, olive). The temperature of the air in the baby's room should be $19-20^{\circ}\text{C}$; for premature infants a room temperature of 22°C is required. If the temperature is lower, the infant (particularly a premature one) should be kept

in a heated crib (rubber bags or just plain bottles, but in the latter case the mother must be warned to cork the bottles securely and to tie the cork to the bottle with a strip of cloth or gauze folded into several layers). As the water in the bottles cools it must be renewed.

MANAGEMENT OF HOSPITAL INSTITUTIONS FOR CHILDREN AND PEDAGOGICAL WORK AMONG SICK CHILDREN

Requirements to modern hospitals for children. As the children that are hospitalised may, although rarely, catch some other kind of infectious disease, it is recommended in building children's hospitals to follow the system of self-contained departments with separate entrances (no more than 30-50 beds to a department).

The wards in children's hospitals must not be too large; the newly hospitalised child should first be placed in a small room (for 2-3 beds), and only after several days, when there are no symptoms of contagious diseases or in the absence of any contact with disease at home, the child may be transferred to a larger ward.

In the larger wards children who cough or are suspected of having influenza or whooping cough, or have pneumonia should be semi-isolated by glassed partitions.

There must be a porch for the children to take airings on in children's hospitals or in pediatric departments of general hospitals.

Practice has shown that diseases such as whooping cough and pneumonia take a more favourable course when the patients spend a large part of their time in the cool open air. Therefore, the windows in the wards must be fitted with special transoms for airing the premises. This is particularly important during sleep; sleep in fresh air is longer and deeper.

As secondary infections are usually introduced from without, it has recently become customary to build so-called quarantine wards where the child spends the first 2-3 weeks in the hospital in an individual ward or cubicle with a closed door and a small entrance hall or ante-cubicle (also with a door) containing a wash-stand, a nurse's white coat and cap. The nurse washes her hands, puts on the coat and cap and only then enters the ward. When she leaves she takes off the coat and cap and again washes her hands. The partitions between the cubicles are of glass, and therefore the nurse sees what is going on in adjacent cubicles. The doors of the ante-cubicles open on a common corridor. All the objects used in caring for the child, his bath, and his lavatory are private. Food is delivered to the cubicle from the corridor through a small service

transom in the wall of the ante-cubicle. This method of boxing children is called individual isolation.

Practice has shown that under this system of hospitalisation an insignificant rate of secondary infections is observed. There are isolation wards constructed on the same lines for children with undefined disorders, only each cubicle has its own entrance from the street (the so-called Meltzer cubicles). Meltzer was a Russian engineer who first proposed isolation wards of this type. If there is no separate building, then part of the department is divided into cubicles and new children are accommodated there for 2-3 weeks. The most important things in the isolation ward system are frequent hand-washing upon entrance and exit of the staff to and from the cubicle, and the wearing of a separate coat in every cubicle.

In order to avoid all manner of infection the most scrupulous cleanliness of the hospital premises is required. The floors must be mopped every day with a caustic solution; the walls, too, must be scrubbed as frequently as possible. Any of the staff with suppurative processes on the skin (furuncles, paronychia) should not be allowed to take care of the children. Nurses or attendants with influenza in a light form must wear gags over their mouths and faces so as not to breathe on the patients. Where there are no isolation wards the staff's hands should always be washed on going from one child to the next.

The temperature of the air in the hospital should be 18-20°C, with no sharp changes. The walls, floors, bath and lavatory of the isolation cubicle are thoroughly scrubbed after the patient has been dismissed. All objects of care for the patient, the crockery and other utensils must be thoroughly boiled.

Establishment of proper conditions in the nursery wards is a factor of great importance. It must be remembered that hospitalisation in itself, the separation of the child from his family, is undoubtedly a source of mental distress to the child. It is therefore necessary to try to make the hospital as comfortable as possible, so that the new ways of hospital life affect the child's mentality as little as possible. With this in view the child should be initiated gradually, from the very moment he is accepted in the reception room, to his new environment. If possible, the mother should be allowed to bathe and dress him on reception, before a stranger takes him away to the ward. If the child is very upset in the first hours in the hospital it is recommended to let his mother or another familiar person visit him. The ward nurses and attendants should show the new patient the utmost attention, distract him by talking and with simple games.

When the children fall asleep at night no bright light should burn in the ward; during the afternoon nap the room should be darkened, if possible. While the children are asleep complete silence must reign. The staff must not engage in loud conversations. A special ward should be set aside for children suffering from disorders that are treated chiefly by means of deep natural sleep (chorea minor, meningitides); in this ward the windows should be curtained, the floor carpeted, and the staff permitted to speak only in whispers. Accommodations must be arranged so that the noisy convalescents are not in the same ward as the severely ill children.

The walls in hospital nursery wards should be of soft, pleasant colours, potted flowers on the window sills, and, if possible, an aquarium, are desirable objects. Simple colourful pictures should hang on the walls.

All measures of personal hygiene should be observed in the care of sick children: hands washed before meals, teeth brushed, etc. Young children should be placed on their potties regularly, if their health permits.

In order to give better care to baby patients it is recommended to invite the mother's help; this is obligatory if the child is breast-fed. There must be special rest-rooms and lunch-rooms for mothers in the children's hospital or department. There must also be sleeping quarters for mothers who live far from the hospital or are not local residents.

Each department of pediatric hospitals must have on its staff a kindergarten and school teacher. The duties of the teacher include the management of the recreation of convalescents (games, lessons, and for the older children suitable reading matter). In departments where children are hospitalised for prolonged periods (for tuberculosis, rheumatism and other chronic diseases) the teachers also teach the children according to the programme for elementary and secondary school.

The presence of teachers in the children's hospital does not exclude pedagogical work on the part of the doctors and nurses. In small departments, where the staff does not include teachers, the entire educational work rests with the medical staff.

CARE OF THE CHILD IN ILLNESS

In no other field of medicine is *good care* of the patient so important as in pediatrics. *Full-value nutrition* is a leading factor in the outcome of the disease. Besides, many gastrointestinal

disorders are treated by means of special diets. Extensive use of fresh air, sun and water (physiotherapy) is also important. Thus, *good care, diet, and physiotherapy* are the basis of treatment in children's diseases.

With the development of *serology* therapeutic serums obtained by immunising horses with toxins or microbes are being used successfully for the treatment of certain disorders (diphtheria, scarlet fever).

Chemotherapy (sulfonamide preparations), antibiotics (penicillin, streptomycin, synthomycin) have in recent years yielded excellent results in pneumonia, meningitis, dysentery, toxic dyspepsia, erysipelas. Vitamin therapy has also opened a wide vista for exploration.

Drug therapy is very limited in childhood, and includes only a small number of preparations (specific and symptomatic).

In caring for sick children *cleanliness of the premises* is quite important; the floor should be mopped with a moist cloth, not swept with a dry broom. Only the persons caring for the child directly should be allowed to enter his room, as otherwise an infection might be introduced. This is why children are so seldom allowed visitors in pediatric hospitals. Exceptions are made for severe cases.

Clothes and bedclothes must be changed frequently, thoroughly washed and ironed. Diapers must never be rinsed off only. A waterproof material (oilcloth, plastic) should be placed under the sheet of a baby or severely ill child to protect the mattress.

The child's clothes should be comfortable, with no superfluous folds, and made of a material that washes easily. In the warm season the clothes should be lighter, so that the child does not become overwarmed. For the newborn, and during the first months of life, white cotton and cotton flannel kimonas are recommended. The diapers should be made of a thicker material (linen, fustian, flannel), 100×100 cm. Before diapering the baby it is dressed in its kimona, and a special smaller diaper, a diaper lining (50×50 cm), made of a soft washable cotton cloth or, still better, of a piece of gauze folded in four is wrapped around its buttocks and groins. (Fig. 13 shows how to diaper babies.)

Hose-and-drawer combinations ("crawlers") are recommended for babies of 3-4 months and older.

The clothes of children over a year old consist of a dress or drawers sewed or buttoned to a waist (Fig. 14). It is desirable that small children wear clothes of soft, pretty colours; if the child is allowed to walk around, he must wear stockings, shoes, a pajama

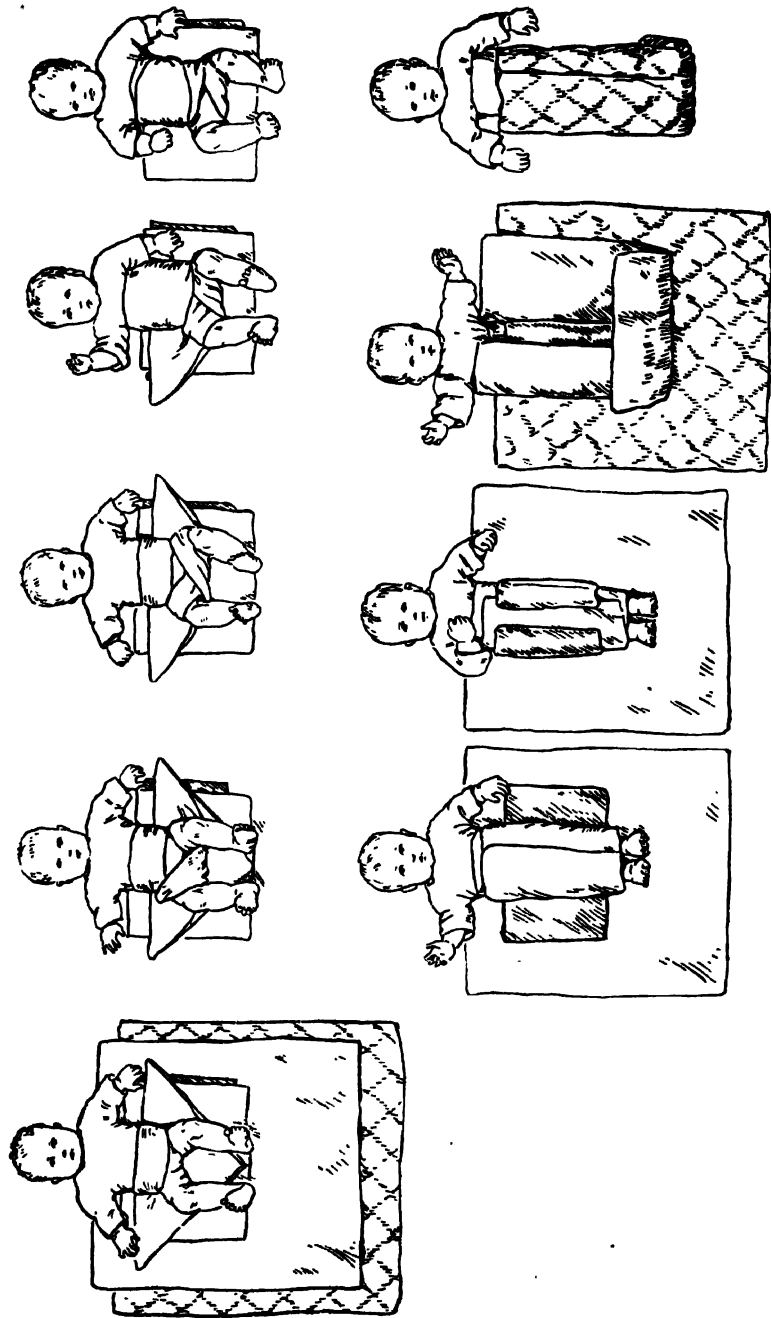


Fig. 13. How to dress an infant

suit or a warm bathrobe. The stockings are fastened to the waist by elastic garters; round garters that compress the leg or thigh, interfering with blood circulation, are forbidden.

In the cold time of the year babies are carried outdoors in special warm sleeping-bags (Fig. 15). Older children must also be warmly dressed in the winter, and if it is necessary for them to be out in the fresh air in their beds they should have extra warm blankets.



Fig. 14. Dress of child over
a year old

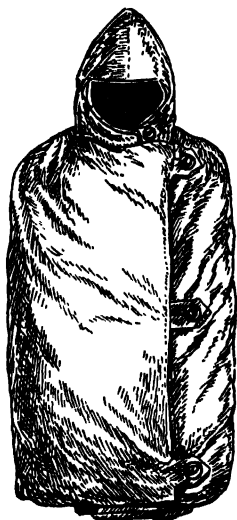


Fig. 15. Warm sleeping-bag

The child should have *his own eating utensils* and also *special articles used in caring for him*. All these objects must be thoroughly washed in water, and boiled (if boilable). On coming home from work parents should wash their hands and change their clothes before they touch their sick child.

When a child is ill his face and hands must still be washed every day, and he must be bathed either every day or every two-three days, as the doctor orders.

If a child is very ill, his face, neck and the area behind his ears are rubbed with a swab of cotton moistened in warm water; first a towel is placed under the head, to protect the pillow. Convalescent children can wash their hands and faces themselves over a basin

brought by a nurse and under her supervision; if the child is too young or too weak to cope with the job, she helps him. Older children may use the wash-stand. Each child should have his own soap, tooth-powder (or tooth-paste), tooth-brush and towel. Every morning when the child wakes up the nurse or attendant makes his bed; older convalescent children do this themselves, except for shaking out the sheets and covers and changing soiled bedclothes for fresh ones.

In changing the bedclothes of severely ill children the patient must not be made to sit up. If he is very young a second nurse holds him in her arms while the bed is being made; an older child may be placed on an adjacent spare bed, or if there are none some other technique is employed. One nurse may put her right arm and hand under the child's body and head, placing her left hand under the small of his back and thus raising him slightly. Meanwhile another nurse or attendant changes the sheet, first wiping off the waterproof under it. Before placing the sheet under the raised child the nurse rolls it up at both sides, then swiftly pushes one end of the roll under the child and unrolls the other end, smoothing the sheet out on both sides.

In changing the pillow-case one nurse lifts the child's head very slightly, while the other changes the pillow-case, or, if necessary, the pillow.

To avoid bedsores severely ill children must be turned from one side to the other from time to time, and they must not be allowed to lie too long on their backs; the sacral region should be sponged daily with 20° alcohol. If the formation of a bedsore is beginning (redness of the skin in the small of the back) the patient should be placed on a rubber ring, the sore place over the hole in the ring. If a bedsore does form the wound should be dressed with a salve (boric acid petroleum jelly, streptocide ointment, Vishnevsky salve) and covered with a gauze napkin.

The doctor prescribes regular hygienic or therapeutic baths for the sick child (see lower). High temperatures dry the lips and make the corners of the mouth crack. Therefore, the child should be offered frequent drinks, and petroleum jelly or lanolin be applied to his lips.

It is necessary to take care of the *mouth* of the sick child. Young children are given sweetened water with fruit juices, while older children are in addition to this given mouth rinses (boiled water or a 20 per cent boric acid solution). The child's teeth should be cleaned every day with a soft brush or cotton tampon, using tooth-paste or powder.

In lesions of the *throat* (pharyngitis, laryngitis, etc.) older children are given various solutions for gargling (rivanol 1:1,000, 1-2 teaspoonfuls per glass of water, potassium permanganate solution, etc.).

The *nose* and *ears* must be looked after. The nose is cleaned daily with a pointed twist of cotton dipped in mineral oil, while for the outer ear and entrance to the ear duct a dry or slightly moistened (in boiled water) twist of cotton is used. If the doctor prescribes *nose drops* they should be instilled with an eye dropper, the patient lying on his back, his head restrained.

Ear drops are instilled with an eye dropper, the child lying on his side; the drops should be warmed before use.

The eyes are wiped with cotton swabs dipped in boiled water, from the outer to the inner corners. If eye drops have been prescribed the child's head is restrained by holding his face with the left hand; the eyelids are opened with the thumb and forefinger, and the drops let in. *Eye salve* is applied with a glass applicator: a blob of the salve the size of a pea is dabbed over the edges of the eyelids.

A *vomiting* child is laid on his side. If it is necessary to ascertain the amount of food ejected in vomiting a clean napkin or diaper (preliminarily weighed) is tied around the patient's neck. After he has thrown up the napkin is again weighed and the weight of the vomitus is determined by subtracting the initial weight of the napkin.

To avoid the irritation of the anal region that accompanies frequent loose movements (diarrhea or dysentery) vaseline (petroleum jelly) should be applied to the anus. An enamel or porcelain (or any other sterilisable) bedpan is placed under the child's buttocks, or several layers of lignin or grey cotton cloth that are later burned together with the feces. After the child has had a movement the entire bedpan area should be sponged carefully with warm water and then vaseline applied to the anus. In prolapse of the rectum (prolapsus ani) the child is placed on his side and the rectum is pushed into place with a vaseline-coated finger, after which the anus is bandaged in the following manner: first gauze and cotton are placed on it, then a gauze belt is tied around the child's abdomen. A strip of gauze is tied to this belt in the back, then it is passed over the anus and between the legs, and fastened to the belt in front. In prolapsus ani the child must in no case whatsoever be permitted to sit on a potty or bedpan; he must defecate lying on his back, into a bedpan or diaper.

The genitalia of *girl patients* should be washed every morning and evening with a swab of cotton dipped in boiled water, from front to back. If the external organs are irritated or red they must be treated with bismuth salve.

We shall now proceed to a description of the different medical procedures employed in the treatment and care of sick children.

Procedure for urine collection in infants. For collecting the urine of a male child the rim of a thick-walled test tube is covered with adhesive tape, or a strip of gauze is tied around it, the penis is inserted into the tube and the latter is taped to the pubic area or

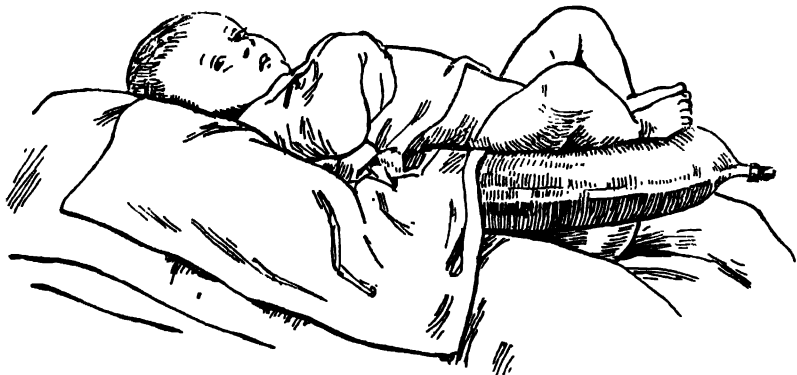


Fig. 16. Collection of urine specimen from female babies

secured by tying the gauze band around the child's body; the tube is inclined downward. The child is restrained in bed by a towel or diapers tied to the sides of the bed. He is given an abundant amount of liquid to drink. The perineal area of female children should be washed *before taking a specimen* in order to ensure that the whites (leukorrheal discharge) that girls sometimes have do not enter the specimen, as this might confuse the result of the urinalysis. To collect urine specimens of female children a wide-lipped glass vessel is used; its rim is covered with adhesive tape and it is taped to the external genitalia. Besides this, a small pan may be placed under the child, and the latter restrained in bed by means of two towels (Fig. 16). If it turns out to be totally impossible to obtain a specimen by these means it is recommended to place a piece of absorbent cotton between the child's legs; after the child has urinated the cotton is squeezed out and the thus obtained specimen is sent to the laboratory. However, part of the urine elements remains in the cotton, and therefore this collection technique does not yield precise results.

Urine specimens may also be obtained by means of a *soft catheter*. The urethral opening of the female child is washed with a solution of potassium permanganate and then the boiled catheter is inserted into the urethra; the urine is collected into a sterile test tube. In male children the insertion of a soft catheter is sometimes not feasible; in such cases a metal catheter of the smallest diameter is employed. It is cautiously inserted into the urethra, bent almost to touch the abdomen, then slowly elevated and turned to the right; the catheter enters the bladder, and urine appears at its opening. Force is not permitted, as it is easy to make a false move.

Taking sputum specimens. Sputum is obtained with a cotton tampon on a metal spatula that is inserted deep into the throat—up to the epiglottis; here the sputum is collected by pressing the spatula against the root of the tongue and thus making the child cough. As children usually swallow sputum, it may also be obtained by gastric lavage on an empty stomach; particles of the sputum are fished out of the lavage waters. The sputum specimen is examined for diplococci, for tubercle bacilli, and other bacteria. It is likewise *recommended to collect fecal specimens* to be examined for tubercle bacilli that are often encountered in the intestines.

Taking specimens of mucus from the throat and nose. Mucus is obtained from the nose and throat with special sterile tampons supplied in test tubes. The tampon, attached by a wire to the cork of the tube, is taken out and carefully passed over the mucous membrane of the tonsils, then just as carefully inserted into the nostrils, care being taken not to injure the mucosa. The specimen obtained is immediately sent to the laboratory for culture and analysis (most frequently for diphtheria bacilli and hemolytic streptococci).

Extraction of Gastric Juice for Analysis, and Gastric Lavage

Gastric juice is extracted after a test breakfast (older children are given a piece of white bread and some tea, infants are given rice water) by means of a stomach tube.

The tube is entered into the mouth, moved to the back of the throat, and after the child starts making swallowing movements the tube is pushed in deeper, without undue pressure, through the esophagus into the stomach; the contents of the stomach are

ejected very soon through the tube. They are collected in a jar and sent to the laboratory. In order to withdraw the contents of the stomach of infants a soft plastic catheter is employed; it is inserted through the mouth or nose and pharyngeal space. Its free end is preliminarily attached by means of a short glass tube, to a 60-70 cm rubber tube.



Fig. 17. Gastric irrigation

To perform gastric lavage (Fig. 17) a glass funnel is attached to the end of the rubber tube. Water, Ringer solution or some other lavaging solution (as prescribed by the doctor) is poured into the elevated funnel, and the liquid passes into the stomach. When the funnel is held beneath the level of the child's head (he is placed on his side on a table, buttocks elevated) the stomach contents pour out together with the lavaging liquid. When the stomach has been emptied the funnel is again elevated and a new portion of the liquid poured in. This procedure is repeated several times until the fluid emerging from the stomach becomes clear. At the end

of the lavaging procedure 50 to 100 ml of Ringer solution are left in the stomach. In *domestic conditions* gastric lavage is performed by giving the child an abundant amount of warm water to drink (1-2 litres), until vomiting is evoked. This procedure may only be employed if the child is conscious and in a condition to swallow.

Taking specimens of feces. The bowel movements of infants are taken by means of a narrow glass tube plugged with a bit of cotton at one end, while its other end is pierced through a cotton plug and placed in an ordinary sterile test tube; after the cotton has been removed the end of the tube is covered with petroleum jelly (vaseline) and inserted into the rectum; it is soon filled with fecalia, then withdrawn and plugged up again with the cotton. The tube is sent to the laboratory to be tested for dysentery, typhoid and *Escherichia coli* bacilli.

Enemas. *Purging enemas* are given with water at 30-35°C, through a rubber syringe with a soft tip, or through an Esmarch can; a piece of soft rubber tubing must cover the hard tip of the latter. The can is hung at a height of a metre above the bed. The child should be lying on his left side. Amounts of water injected: for the newborn 50 ml, in the first six months of life 100 ml, in the second six months 150 ml, from one to two years 200 ml, from two to six years 300 ml, from six to ten years 400 ml.

High enemas are given by means of a can with a long rubber tube, the tip of the latter is inserted beyond the second sphincter ani muscle. The child must lie on his left side.

Hot-water enemas (for retaining water). The water is heated to 39-40°C, and is injected very slowly. 50 to 100 ml of water (or Ringer solution) may be given.

Oil enemas for loosening the bowels are made by injecting 30 to 100 ml of warm boiled vegetable oil into the rectum by means of a rubber syringe. To retain the enema, the anus of young children is plugged with a swab of cotton, and his buttocks are tightly compressed for about ten minutes. If the enema has no effect, an hour later an ordinary water (purging) enema may be given.

Glycerin enemas are given either with pure glycerin (1-2 table-spoonfuls), or with one teaspoonful of glycerine added to the water in the enema bag or can.

Medicinal enemas are given in a similar manner, only with the addition of the corresponding medicines.

Nutritive enemas are injected three to five times a day, 50 to 100 ml at 40°C; the child should be placed on a pillow on his left side, his buttocks elevated. After the enema has been given the

anus is plugged with a piece of cotton and the buttocks are pressed together for five to ten minutes. Nutritive enemas are made with fresh cream (50 to 100 ml + 1 tablespoonful of sugar); with milk (50 to 100 ml + 1 egg yolk and one to two teaspoonfuls of sugar, or two g of peptone); with glucose in normal saline solution, or with glucose in breast milk. A purging enema must be given 30 minutes before the nutritive enema.

Sedative enemas for treating convulsions contain chloral hydrate dissolved in water, heated to 40°C, given after a purging enema; 35 to 50 ml of a 2 per cent solution is slowly injected by means of a rubber syringe; and then the buttocks are pressed together for 10 minutes (so that the liquid does not flow out).

Drip clysis is employed for the slow absorption of replacement fluids in dehydration of the system; it is given from an Esmarch can with a soft tip. The soft tip is taped to the anus and a glass tube is mounted below the spigot of the tube leading from the can. Thus visual observation is provided for the drip of the fluid to the rectum, and it is possible to regulate its rate. To prevent cooling of the fluid a hot-water bag (45°C) is placed on the rubber tube near its tip. The Esmarch is hung at a height of one metre above the patient's bed. The usual replacement fluid is Ringer solution (100 to 200 ml), given for one or two hours.

Test punctures. Pleural puncture. (Thoracocentesis.) An assistant immobilises the child in a sitting position, the bad shoulder elevated. The skin is swabbed thoroughly with a 7.5 per cent alcoholic solution of tannin. Usually a thick hypodermic needle on a 5 g syringe is employed; it is inserted dorsally on the scapular line in the seventh-eighth intercostal space just above the upper margin of the lower rib, or laterally on the axillary line in the sixth-seventh intercostal space. When the needle enters the pleura the fluid is aspirated. If upon aspiration the barrel of the syringe recedes (negative pressure), it means that there is no liquid in the pleural cavity; air bubbles appear if the needle enters the lung. When the pleural fluid has been obtained the needle is quickly withdrawn and the skin pinched to prevent air from entering the pleural cavity. The site of the puncture is sealed with cotton and collodium. The aspirated fluid is sent to the laboratory in a sterile test tube, to be examined for diplococci, streptococci, and other bacteria. If there is an abundant amount of liquid it is withdrawn in several stages with the same syringe, or a special apparatus is employed.

Obtaining spinal fluid. Lumbar puncture. The child lies on his right side, his neck strongly flexed and knees drawn to the ab-

domen. As the spinal cord terminates at the level of the first lumbar vertebra, punctures may be performed beginning with vertebra II without fear of injuring the spinal cord; the line that connects the iliac crests and passes through the middle of the IIIrd sacral vertebra may serve as a point of identification. The puncture is made strictly on the median line, the needle-point being inserted closer to the lower vertebra, slanting slightly upward. The skin of the back should be disinfected preliminarily with iodine or alcohol. At the end of the procedure the needle is removed swiftly and the site of puncture on the skin is sealed with cotton and collodium. The withdrawn fluid is collected in a sterile test tube and sent to the laboratory for testing (protein reactions, cytodagnosis, tubercle bacilli, meningococci, etc.). The test tube should be wrapped in cotton to avoid cooling the spinal fluid, as the meningococci decompose quickly in the cold and do not grow in cultures.

Injections. Subcutaneous injections of medicines are given from sterilised (by boiling) 1 g syringes. The drug is drawn into the syringe from the phial or ampoule, then the needle is pointed upwards and the air that may have been sucked in is ejected. Subcutaneous injections are made on the outer aspect of an arm or thigh, after disinfecting the skin with alcohol. The skin is pinched with the left hand, the right inserts the needle into the base of the fold of skin. When the skin "gives", and it is felt that the needle has entered the subdermal tissue, the right forefinger presses the barrel of the syringe, and the medicine is injected into the tissue. If the skin pales upon injection it means that the liquid is entering the skin; the needle must then be pushed in deeper. After the drug has been injected the needle is withdrawn quickly and the site of puncture is sealed with a piece of cotton dipped in collodium.

Subcutaneous infusions. Warmed Ringer solution* is infused into the subcutaneous connective tissue of the outer aspect of the thigh, 50-200 ml per dose, depending on the child's age; the skin is first disinfected with alcohol. Infusions may be given with the Bobrov apparatus, or with a large syringe (100-150 ml capacity), very slowly.

The Bobrov apparatus is a cylindric graduated vessel; it is closed with a rubber plug, pierced by two glass tubes, bent at right angles; the shorter tube is attached to a rubber syringe for pumping air, the longer to a narrow rubber tube with a needle on its

Components of Ringer solution: Natrii chlorati 7.0
Kalii chlorati 0.12
Calcii chlorati 0.24
Aq. destill. 1000.0

tip. The skin is swabbed with alcohol and the sterile needle plunged through the skin to the subdermal layer. Air is pumped into the vessel and this causes the warm Ringer solution to flow out into the tissue at the point of the needle. The site of injection is aseptically sealed after the needle has been withdrawn. Besides Ringer solution, infusions are also given with 50 to 100 ml of a 5 per cent glucose solution.

When a large hypodermic is used for infusions a boiled rubber tube is fitted over its tip, the Ringer solution is drawn in, and a sterile needle is attached to the end of the tube; the solution is injected into the subcutaneous tissues in the usual manner. If no Bobrov apparatus or large hypodermic are available, the infusion may be given with an ordinary 10 ml hypodermic syringe: the warm Ringer solution is poured into a sterile glass, drawn into the syringe and immediately injected into the tissue; the syringe alone is removed (the needle is tightly plugged with sterile cotton), warm Ringer is again drawn into it, the cotton plug of the needle removed, and the solution injected. This procedure is repeated until the necessary quantity of Ringer solution has been injected.

Moreover, subcutaneous infusions may likewise be given through a sterilised glass funnel to which a rubber tube has been fitted. A hypodermic needle is attached to the other end of the tube. When the warm Ringer solution, poured into the funnel, leaks out of the needle (by gravity), the latter is inserted into the subcutaneous layer; the funnel is held at a height of 1 m above the bed, and is covered with a sterile gauze napkin.

Intramuscular infusions. This type of infusion is given with a 10 g hypodermic syringe, into the anterior aspect of the mid-thigh. It acts much sooner than subcutaneous infusions. The skin is disinfected with alcohol, and then the needle of the hypodermic is plunged deep into the muscle; the liquid is infused by slowly applying pressure to the barrel of the syringe. The liquids usually thus injected are various *therapeutic serums* (against diphtheria, scarlet fever), *antibiotics* and *maternal blood*. Before the injection the ampoule containing the preparation is heated in warm water, then wrapped in sterile cotton or gauze and its tip is broken off by striking it sharply with a knife; the serum is then drawn into the syringe through a needle. The syringe is lifted, needle upward, to eject the air that may have been drawn in, and the injection is given.

Intravenous infusions. Intravenous infusions are given to very young children in toxicoses (toxic dyspepsy, dysentery, etc.). The infusion fluids are glucose and blood plasma.

In certain cases the blood of a donor is transfused, after the blood group has first been ascertained. No more than 5-10 ml of blood per kg of weight is permissible. The site of transfusion is more commonly an antecubital vein or a scalp vein; the skin is disinfected in the usual manner. If it is impossible to pierce the vein under the skin, it is exposed and isolated surgically (cut-down intravenous).

Injection of drugs into the spinal cord. A lumbar puncture is performed and the spinal fluid is let out, then a certain dose (prescribed by the doctor) of diluted penicillin (in pyogenic meningitis) or streptomycin (in tuberculous meningitis) is injected. A number of other drugs are also used in this manner.

Obtaining blood specimens. a) Venipuncture from the antecubital vein: a tourniquet is applied to the upper arm under moderate pressure. The skin is disinfected and the needle is inserted into the vein, pointing shoulderwise; the blood is either drawn into the syringe, or it flows out spontaneously into a graduated vessel held under the needle.

The technique of scalp vein puncture is similar to antecubital puncture. After the operation the site of puncture must be sealed (cotton and collodium); it is advisable to bandage the sealed site (sterile gauze dressing) for a day or two. Venipuncture is performed for both diagnostic (Wasserman reaction, bacteriological culture of blood in bouillon), and therapeutic purposes (in uremia). b) Blood drawn from the ball of a finger: the disinfected skin is punctured with a special or ordinary needle; the blood that flows out is taken for various blood tests. c) Blood from the heel: if blood cannot be obtained from the vein it is taken from the heel by the following technique: the skin is disinfected with alcohol, and a V-shaped incision is made with a scalpel (open side of V upward); the blood that flows out is caught in a test tube (3-5 ml).

Oxygen respiration. Oxygen is given to premature infants who have pneumonia or cardiac disorders. The oxygen bags should be prepared beforehand (two hours) and kept in a warm room. A clean pillow-case is put on the bag, and the respirating funnel is washed in warm water. The funnel is placed 1-3 cm from the mouth of the infant, the stopcock is opened cautiously, and the bag is pressed gently with the hand, so that a small stream of oxygen slowly emerges. Oxygen is given over one-hour intervals for 2-3 minutes. From one to four bagfuls may be given in 24 hours. However, one must never forget that thorough airing of the room and taking the child outside may often be an adequate substitute for the inhalation of oxygen.

Artificial respiration. In asphyxia or deep syncope artificial respiration is indicated. The child's mouth is opened, his throat is swabbed of mucus (with a cotton-swabbed finger), and the tongue is drawn out, then the child's arms are raised to his head and brought down to the chest, flexed at the elbows, the operator gently pressing on the chest with his hands (for the newborn Schulze's method should be avoided, as it may evoke internal hemorrhage).

The Sokolov method is also used quite successfully. The child lies supine, the operator flexing its neck strongly by placing a hand under the back of the head, and simultaneously forcing the flexed knees to the abdomen, then straightening out head and legs. The child is also given a subcutaneous injection of a solution of camphor or caffeine, and oxygen to inhale. It is advisable to carry him out into the fresh air.

Heaters. In cardiac weakness heating devices are put to the feet (rubber bags filled with hot water at 45-50°C, or ordinary wine bottles securely plugged and tied). To avoid burns the bags or bottles are wrapped in a towel or placed in a special cover. Hot sand, heated in a pan, is also a good heating agent that may be used instead of water. Electric heating pads are also widely used.

Application of cold. A rubber ice bag is filled with cold water, or packed with snow or ice and placed on the head of a patient running a high temperature, or on other parts of the body to alleviate pain. Before screwing in the cap of the bag the latter should be pressed to let out air. The ice bag is wrapped in a thin towel and tied to the headboard of the bed.

Massage. Massage is widely employed for the treatment of various diseases of childhood (hypertrophy, rickets, infantile paralysis, constipation, etc.).

Gymnastics. Gymnastics for infants are used in hypertrophy and rickets, and in older children in the treatment of various paralyses and ankylosis. *Respiratory gymnastics* are employed in the after-treatment of pleurisy and other pulmonary afflictions.

The Pirquet test. The surface of the inner aspect of the forearm is cleansed with ether and permitted to dry, then a drop of pure tuberculin is placed on the skin. The skin is abraded at a distance of 10 cm from this drop with a special platinum Pirquet scarifier (by rotationary movement); this is the dry control. Then a similar scarification is performed in the drop of tuberculin. The scarification must be very light, as for vaccination, avoiding appearance of blood. If no special scarifier is available, a vaccination lancet or the dull edge of a scalpel may be used. In 24 hours, or even earlier, a papule appears on the surface of the skin. According to Soviet

medical instructions, the reaction is considered positive in the presence of a papule and redness over a 5 mm diameter.

Mantoux test. This is an intradermal tuberculin test that is now used quite widely. Its technique is more complex than the preceding one, and it calls for a certain amount of skill. The necessary equipment is a thin (if possible, platinum) needle sterilised by flame, and a precisely graduated tuberculin hypodermic syringe. Rigid aseptic precautions are necessary. The needle is inserted into the skin (but not under it!) of the arm or forearm, almost parallel to its surface, and one graduation of a definite solution of tuberculin is injected (a small bleb, 4-5 mm in diameter, should appear and vanish after several minutes). In 24 hours a red papule rises on the surface of the skin at the site of injection. The reaction is considered positive if the diameter of the papule ranges from 8 to 20 mm.

The strength of the tuberculin solutions employed consecutively in the Mantoux test are: 1 : 1,000; 1 : 10,000, etc. The Mantoux test is more sensitive than the Pirquet test.

UTILISATION OF NATURAL FACTORS (SUN, AIR, WATER) IN THE TREATMENT OF CHILDREN. METHODS OF PHYSIOTHERAPY MOST WIDELY USED

Utmost utilisation of the benefits of fresh air, sunlight and water are an important factor in hardening children, and also in treating them in illness. The room of a sick child should be aired thoroughly several times a day. Small transoms are not enough for this. Before airing the room the patient should be covered with a second blanket, and left in the room. His face must not be covered. It is now usual to carry pneumonia patients out into the fresh air for one to three hours even in the winter; pale, cyanotic children breathe easier in the fresh air and fall asleep quietly. In prolonged cases of influenza the child must be taken out of doors; coughs and subfebrile temperatures disappear much sooner then. In the summer a sick child should have fresh air all the time, windows in hospital wards should be opened wide. In the winter the child should be wrapped up warmly and the window kept open an hour or two every day, depending on the temperature outside. Sick children may be carried outside in the evening, too. We know how favourably tuberculous children in sanatoriums are affected by prolonged exposure to fresh air; in children's sanatoriums the patients are kept on porches almost all day, in special warm sleeping-bags.

Air baths are given for treating a number of children's diseases, such, for instance, as bedsores in infancy, various skin troubles at an older age. Air baths are taken in the open air in the summer, and in warm premises in the winter. The child lies naked on his bed for five to thirty minutes. Air baths may be repeated several times a day. *Hot air baths* (under electric lamps) are employed to induce perspiration (sudoripic) in nephritis. The old, harmful superstition—the fear of catching cold—should be combated actively. It is very beneficial for a sick child to be kept in the country, where the air is less contaminated with soot and dust than in the city.

The next important agent in treating children in illness is *sunlight*. Pathogenic microbes are killed by bright sunlight in several minutes; therefore, there should be no curtains on the windows of a sickroom. Even when the child has the measles the room should not be darkened; if the light irritates him his bed should be placed so that the light does not fall directly in his face.

Sun baths are beneficial in the treatment of rickets, tuberculosis and other diseases. The highest intensity of ultra-violet rays is observed in the mountains; therefore, mountain country is a most suitable place for the treatment of tuberculosis and other diseases.

Sunning must be strictly dosed, and the children examined carefully before prescribing sun baths. The period of exposure depends on the intensity of the rays in different times of the day and year, and on the age of the child and his sunlight tolerance. While in the sun the child should wear a white panama hat; the duration of the procedure ranges from five to thirty minutes, and the child must lie first on a side, then face down, then on his back. The time of exposure is lengthened, depending on the general reaction and health. Sun baths may be taken several times a day, but the cumulative daily exposure should not exceed thirty minutes. Particular caution is required in infancy, care being taken not to overheat the baby. After the sun bath the child is rubbed down with a wet cloth, or water at 34-35°C is poured over him (or he is given a quick shower), and then he is dried, dressed, and kept in the shade. If the child becomes thirsty during the sun bath he should be given a drink of boiled water.

In the winter artificial sources of light are employed—mercury vapour lamps emitting ultraviolet rays. These lamps are used for local and general irradiation of the child's body in rickets and glandular tuberculosis. The naked child, wearing blue glasses, is placed on a clean sheet, the lamp is fixed at a distance of 60-100 cm from his body.

Irradiation time ranges from three to fifteen minutes; the procedures are prescribed on alternate days, 15 to 25 in all. If caution in dosage is not observed a redness of the skin may appear; this redness will disappear soon after an oily ointment or salve has been applied to it.

Sollux is a 1,000 cd. electric lamp emitting a high amount of heat rays; it is employed as a pain-alleviating and resolving agent.

Blue light is a lamp that is used in glandular inflammations and in pneumonia for ten minutes three times a day; it is a pain-alleviating and resolving agent.

Diathermy is a form of phototherapy employed for the treatment of infantile paralysis (applied to the spine), in pneumonia (to the chest) and in dysentery (to the abdomen) for three to five minutes on ten to fifteen consecutive days. The current should be dosed carefully in order to avoid burns.

Ultrashort waves (super-high frequencies) are successfully used in the treatment of insidious mastoiditis and pneumonia.

Galvanisation and faradisation. Galvanic and faradic currents are used for follow-up treatment of poliomyelitis, in nocturnal enuresis (bed-wetting), and for other disorders.

The third important natural factor is water. All patients (even patients afflicted with skin diseases) should be given warm hygienic baths (at 35-37°C); in sharp declines of cardiac activity the baths are temporarily discontinued. Infants are bathed every day or on alternate days, older children are bathed according to the doctor's orders, but not less frequently than every five days. Some parents do not bathe their children for months on end because of sub-febrile temperature (37.2-37.3°); however, such disorders are much sooner overcome by fresh air and extensive employment of water procedures. Besides hygienic baths, there are a number of therapeutic baths that are given at different temperatures. Thus, *warm baths* (36-38°C) are given for renal inflammations; *hot baths* (38-40°C) for 5-10 minutes daily are good therapy for pneumonia, pyelitis and epidemic meningitis. The child is immersed in the bath, and then the temperature of the water is raised from 37 to 40°C; a bag of cold water or ice is kept on the patient's head. Another way of giving such a bath is to cover the tub with a large sheet, tying its ends together under the tub. Thus something like a hammock is obtained, and the child is put into it, head raised, body immersed in water. Slow pulses call for cardiac drugs before and after the bath (caffeine, camphor).

General hot mustard baths. 100 g of dry mustard is mixed into two pails of water heated to 37-40°C; the duration of the mustard

bath is ten minutes; it is taken lying down. The bath is covered with a sheet, the ends of which are tied around the child's neck to avoid inhalation of the mustard fumes. After ten minutes the child is rinsed off with clear water. Mustard baths are given as treatment for pneumonia. Besides general mustard baths, *mustard foot baths* are given for false croup; to one pail of water at 40°C one tablespoonful of mustard is added; the bare feet of the warmly dressed child are immersed in the water for ten minutes, then washed in clear water, rubbed with oil, and covered with warm stockings. Besides mustard foot baths, plain hot water foot baths (40°C) are given for false croup.

A number of medicated baths are prescribed for various ailments.

Salt-water baths are employed mostly for rickets. 100 g of table or sea water salt are dissolved to a pail of water. Sea salt should be dissolved in a pan of hot water two hours before the bath, and the solution later poured into the bathwater. The temperature of salt baths should be 34-35°C, their duration ten minutes. These baths are given every other evening; after the bath the child is rinsed off with fresh water. On warm days salt baths may be given in the daytime out in the sun. The total number of baths prescribed is 30 to 40. It is best to give these baths in the summer.

Baths with potassium permanganate are given daily, for treating pyodermic manifestations and all manner of suppurative skin lesions. The duration of the bath is five to ten minutes, its temperature 37 to 40°C. After the bath the child is washed down with clear water. Usually four crystals of potassium permanganate are sufficient for a bath containing two pails of water (a weak pink colour).

Effervescent baths (with carbon dioxide) are given to children rarely, only in heart diseases. Natural effervescent baths may be had in the Soviet Union in Kislovodsk (the Caucasus) from the famous Narzan springs. The patients lie in these baths for 5-10 minutes in a horizontal position. The water should reach only the middle of the chest (the nipples); the child's head must be elevated, as otherwise he may inhale too much carbon dioxide and lose consciousness. The total number of baths prescribed is twelve, every other day. Besides natural effervescent baths *artificial effervescent baths* are in use; carbon dioxide cylinders are employed, or else special sodium carbonate plates are dissolved in the bath water, and then a hydrochloric acid solution is slowly added to the water. The temperature of the bath is 30-35°C.

Sulfur baths. Natural sulfur baths are taken in Sernovodsk, Pyatigorsk, and Matsesta in the Soviet Union. These baths are prescribed for various skin diseases and also for rheumatism. The course of treatment includes twelve baths, five to ten minutes each. For artificial sulfur baths 60 to 80 g of flowers of sulfur (sublimed sulfur) are added to 30 litres of water. The temperature of the bath should be 30-35°C.

Baths with pine extract. Prescribed for nervous children. One-half to one teaspoonful of pine extract or powder is dissolved to a pail of water (35°C); the duration of the bath is 10-15 minutes. After the bath the child is washed down with clear water.

Hot packs are employed for inducing perspiration. A woolen blanket is spread over the bed, then a waterproof sheet (plastic, oilcloth, etc.); a sheet dipped in hot water covers the waterproof, and the naked child is quickly wrapped up in the wet sheet, the waterproof, and the blanket. After 20 to 30 minutes he is unwrapped, dried quickly, dressed warmly, and covered up.

Mustard packs are made as follows: a warm blanket covered with a dry diaper is spread over the bed; a second diaper, soaked in hot mustard water (two tablespoonfuls of mustard to two glasses of water) is placed over the first. The child is swiftly wrapped in the diapers and blanket and kept so for 20 to 30 minutes, then sponged down with warm water, dried, dressed in warm clothing and covered with a warm blanket.

Mustard poultices are used in the treatment of pneumonia. One tablespoonful of pure mustard or half mustard, half flour (for young children) is mixed with 2-4 tablespoonfuls of warm water; the resultant paste is spread over a handkerchief or napkin, covered with another piece of thin cloth (another handkerchief) and placed on the chest or back of the child for ten minutes. The reddened area of skin is sponged off with warm water, and some boiled vegetable oil or fresh butter is rubbed into it.

Fomentations are made with linseed, wheat farina or oat flour. Two small linen bags are filled with linseed (or another fomenting substance) and immersed in a kettle of water boiling on the stove; when the seed has been cooked soft, one bag is taken out, slightly cooled, placed on the site of pain (oil should first be applied to the skin), and covered with a piece of flannel; when the bag gets cold it is replaced by the second one, while the first is again immersed in the boiling water. Fomentations are applied several times a day for ten minutes.

Warming compresses are used for treating inflammations of lymph nodes, phlegmons, digestive disorders, etc. A clean cloth or

gauze napkin, folded in half, is soaked in hot water, wrung out and placed on the site indicated by the doctor. The wet cloth is covered with waterproof material or wax-paper and a layer of cotton wool or flannel (larger than the waterproof material), and all this is bandaged securely or tied in place by strips of cloth sewed on to the piece of flannel.

Compresses may be replaced by dry warmth, hot-water bottles, or a blue light lamp for ten minutes three times a day. Instead of water, a 15 per cent solution of alcohol is sometimes used for a compress, or, in skin irritations, boiled vegetable oil.

Salt-water body sponges are given either in the morning or evening. A teaspoonful of table salt is dissolved in one glass of water; the temperature of the water is gradually lowered from 35° to 25°C. The body is sponged part by part, each part dried, and then the whole body is rubbed down vigorously with a turkish towel.

Neurotic children are also given *warm shower baths* for 5-10 minutes, the temperature of the water being gradually lowered: if no shower installation is available a simple watering can for sprinkling flowers may be used. After the shower the child is rubbed dry with a turkish towel.

Proper care is of paramount importance in the treatment of sick children, therefore, hospitalisation is best for severely ill children.

Besides hospitals, our country possesses a network of sanatoriums for children, where they are given highly nourishing food and spend much time in the fresh air.

DRUG THERAPY

The administration of drugs in childhood should be restricted as far as possible. Routes of administration are oral, parenteral (subcutaneous, intramuscular, intravenous injections), by lumbar puncture (injections into the spinal column), rectal, by application to the mucous membranes, and by inhalation.

It is advisable to give internal medicines in solutions, mixtures and drops. Mixtures are flavoured and sweetened with berry or fruit syrups. Decoctions and infusions spoil quickly, particularly in the summer, losing their therapeutic effect; they should therefore be prescribed for no longer than three or four days running.

Fluids for oral administration are measured in spoonfuls (teaspoons, dessertspoons, tablespoons), or in drops. One tablespoonful holds 15 ml, one dessertspoonful 8 ml, and one teaspoonful 4 ml of fluid medicine. The weight of a certain number of drops of medicine varies with the medicine: there are 20 drops in one

ml of water, or diluted acid and alkaline solutions, 40 drops in one ml of vegetable oil, and 50 drops in one ml of alcoholic tinctures.

Powders should be given in small portions of sweetened water; they may only be mixed into the child's food (cereals, puddings, soups) if they possess no unpleasant taste or odour; otherwise the very look of the food in which the medicine was given may later on disgust the child, and he may refuse to eat it when there is no longer any medicine in it.

Pills or tablets are only for older children, not for babies.

Pediatric drug dosages. The Soviet pharmacopeia recommends a table, based on age, for determining drug dosages for children (the adult dose is the full unit).

Age in years	0 to 1	2	3	1	5	8	12
Dose	1/40 to 1/12	1/10	1/8	1/6	1/4	1/3	1/2

For computing dosages for babies younger than one year, the following table based on months of life has been proposed by Repkin. Its values are expressed in per cents of adult doses.

Age in months	0 to 3	4 to 6	7 to 9	10 to 12
Dose	2.5%	5%	7.5%	10%

The above tables are useful for prescribing the majority of pediatric medicines except cardiac drugs and chemotherapeutic agents. Dosages of these drugs are based on the anatomic and physiological features of the growing organism; they were established by clinical observations of their efficiency and of the tolerance of the system at different age levels. It was shown that the dosages tabled above were not adequate for obtaining optimal therapeutic effects with these preparations, and that they had to be increased significantly.

The following table is a good guide for determining the dose of cardiac medicines; the adult dose is taken as a whole unit. The table was first proposed by Professor Troitsky.

Age	1 to 2 months	3 to 4 months	7 to 9 months	10 to 12 months	2 to 3 years	4 to 7 years	8 to 9 years	10 to 14 years
Dose	0.1	0.2	0.25	0.3	0.4-0.6	0.7-0.75	0.8-0.85	0.7

An appendix to this book contains tables of the various types of pediatric drugs (Tables 1-11).

MORBIDITY IN CHILDHOOD AND GENERAL AND SPECIFIC PROPHYLAXIS

Childhood is a most important phase of human life, covering a period from birth to 15 years. The separate periods of childhood differ greatly in their biological characteristics. Thus, these characteristics make the child to a certain extent dependent on *environment and determine the types of morbidity and causes of mortality* at the different stages of childhood.

From the moment of birth to the 15th-20th day of life the child is called *newborn*. The principal physiological characteristic of the newborn infant is that it still possesses many features of the *pre-natal* or *fetal* period. The skin of the newborn is very delicate, its alimentary tract differs significantly from subsequent phases in activity and microbial flora, the heart, lungs and endocrines are functionally immature, the functions of many organs and of the nervous system are undeveloped, heat exchange is very unstable; all this makes *the newborn very sensitive to external* harmful influences. Moreover, the very act of birth is quite a serious moment for the baby, and not infrequently complicated deliveries are the cause of future disorders. Thus *types of diseases* of the newborn and causes of mortality at this stage of life are to be understood.

Diseases of the newborn are: 1) developmental defects, remnants of embryonic (fetal) processes (deformities); 2) injuries afflicted during delivery (birth injuries), such as brain hemorrhages, dislocations, fractures, etc.; 3) congenital diseases (syphilis and others); 4) infections contracted by the infant during birth (tetanus of the newborn, gonorrhea, sepsis). All these diseases are factors in the mortality rate of the newborn.

The younger the infant, the higher the mortality rate; it is at its highest during the *first days and hours of life*. Hence the principal measures for lowering newborn mortality are improvement of parents' health, proper management of obstetric services, good care of the newborn infant, and breast milk nutrition.

The second stage of childhood is infancy (to the end of the first year). In this stage—as in no other—does the most *intensive* growth occur. During the first year of life the child *trebles* his weight. For this a comparatively large amount of physiologically *suitable* food is required—*breast milk*. The chief causes of disease and death in this period of life are due to the *intensive* activity of the digestive system and its dysfunctions.

Diseases of the digestive system are the disorders most frequent in infancy.

The chief cause of gastrointestinal disorders at this age is deprivation of breast milk and infections that may be transmitted in artificial feeding. The infant mortality rate is highest among *bottle-fed babies*.

Bacteriological contamination of cow's milk, its inadequate chemical values, inferior development of babies nursed exclusively on formulas (more frequent occurrence of rickets) are all factors conducive to a higher frequency of gastrointestinal diseases among infants deprived of breast milk, and their higher mortality rate. These factors define *measures for avoiding diseases*: encouragement of breast-feeding, provision of opportunity to breast-feed, instructing mothers in baby care, correct management of mixed feeding. All these measures are widely enacted in the Soviet Union on the basis of Soviet legislation; Soviet law guarantees mothers maternity leave two months before and two months after delivery, and provides time for breast-feeding during the whole period of lactation. Infant mortality decreased significantly within a short time after the October Revolution. Therapeutic and prophylactic children's institutions, associations of hospitals, polyclinics and child welfare centres (day nurseries, infant health centres, child homes, etc.) are the most active proponents of the above measures.

The *second place* in frequency and severity at this age is taken by *respiratory diseases*. Owing to its specific structural features the respiratory tract is easily accessible to external influences: air-borne infections (the pathogens of influenza, measles, whooping cough) easily enter the organism through the trachea, bronchi and lungs.

Prophylaxis of diseases of the respiratory tract includes the wide propagation of hygienic rules: prolonged exposure of children to fresh air throughout the year, cleanliness of the child's body, dress and underwear, of the premises he frequents, protection of children against contacts with sick persons.

Between one and three years (nursery age) and three to seven (preschool age) the child gradually becomes accustomed to ordinary nutrition. The nature of his food changes, rates of height and weight gains slow down. However, the digestive and respiratory systems are still easily affected by external agents. Therefore, *intestinal and respiratory disorders and resultant mortality* are still high in this period and should be actively combated. This is also the age when the harmful influences the child was exposed

to in infancy may display themselves: deprivation of breast milk, erratic feeding, bad care—all factors conducive to rickets, tetany, dystrophy, blood formation disorders, chronic infections (tuberculosis).

Moreover, another specific feature connected with a new danger is manifested at this period. In the first 3-5 months of life babies hardly ever catch acute infections (measles, scarlet fever); this is due to the greater *isolation* of babies, as well as to the fact that the infant receives from his mother, through the *placenta* and *breast milk*, antibodies that protect him against contagious diseases. The nonsusceptibility of infants to infections in the first months of life is also due to the fact that breast feeding ensures the best development of children. After one year of age this temporary immunity gradually disappears, the child comes into closer and closer contact with his environment (crawling, walking, dirty hands and transmission of infections to mouth, greater opportunity for contacting infection-carriers).

As a result, children begin catching infectious diseases at the age of one to two years, and by three to six years the *morbidity rate* goes up.

The diseases most frequent at this age are *measles*, *scarlet fever*, *diphtheria*, *whooping cough*, *chickenpox* and other *acute infectious diseases*; they are the main causes of mortality at this period of life.

Early childhood is likewise afflicted by a group of diseases connected with bad care and negligence. They include injuries (traumas), particularly in large cities (traffic accidents), *poisoning*, *burns*. In cities children often are poisoned by swallowing *caustic alkalis* (*caustic soda*, for instance); often the outcome is fatal, or grave deformities remain. The population should be acquainted with precautions for keeping such substances.

Child morbidity and mortality may be overcome chiefly by *improving the child's living conditions*, by propagating and explaining rules of baby and child hygiene and care. In this respect there are unlimited opportunities in the Soviet Union, thanks to the great improvement of the popular welfare and healthier conditions of work and life. The part child-care institutions play in the enactment of these measures is immense.

Besides measures of a *general nature*, it is imperative to advance *specific prophylaxis* on a wide scale: encouragement and conduction of *prophylactic immunisation against smallpox, diphtheria, measles*. The proper enactment of these measures at an *early age is a guarantee of their greatest efficiency*.

The school age (seven to fifteen years) is a period of gradual maturation and development, during which the child's nervous system and organs of sense attain their final development. In the later stage of this period the sex glands become active, and this is accompanied by a height spurt and intensive development of the vascular and endocrine systems. Susceptibility of communicable diseases decreases in this period; however, diseases of the *cardiovascular* and *nervous systems*, of the *endocrines* and *hematopoietic* (blood-forming) organs are observed. A comparatively large place is taken by *chronic infections* (tuberculosis and rheumatism).

Measures for preventing diseases among this age group are the same as described above. Prophylactic and other children's institutions are a great health-promoting factor (open-air schools, playgrounds, summer camps, summer sanatorium colonies, special out-patient clinics [dispensaries]).

In addition to the enumerated, there are specific measures enacted on a wide scale—immunising inoculations.

The antituberculosis drive is based on the extensive activities of special *antituberculosis organisations*, the mainspring of which is the *dispensary*, a specialised out-patient clinic where tuberculous children and children suspected of having this disease are regularly examined and treated. Another obligatory measure is the inoculation of newborn infants with vaccines derived from cultures of weakened tubercle bacilli (BCG—*Bacillus Calmette-Guérin*).

Specialised clinics, polyclinics, and malaria stations wage an organised drive against malaria. In recent years a similar planned offensive has been launched against rheumatism by the institution of specialised pediatric rheumatism offices in the ordinary polyclinics. This work is united and sponsored by the Children's Section of the All-Union Rheumatism Board.

DISEASES OF INFANCY

DISEASES OF THE NEWBORN

Congenital Anomalies and Deformities

Various deviations from normal may occur in the structure of the different organs during the prenatal development of the fetus. Moderate deviations (developmental anomalies) may have no significant effect on the function of the organ; however, more marked deviations may malform the organ, and even the system as a whole, greatly affecting the child's subsequent general physical and mental development. The cause of this or that defect or deformity is a faulty formation of the organs or parts of the embryo; this, in its turn, is due to an infectious or toxic maternal disease or to a disease of the fetus itself.

A congenital disorder of the *skin* sometimes observed is *telangiectasia*, manifested by dark red spots due to the dilation of groups of capillaries, usually located in the back of the neck or on the bridge of the nose.

Often these spots become visible only when the baby cries loudly—"angry spots".

Other instances of vascular anomalies are vascular tumours,—*angiomas* (*naevus vasculosus*) (so-called strawberry marks) noticeable at all times. These tumours often grow with time. Angiomas occur on all areas of the skin: the face (sometimes large purple-red marks) or the body (small multiple formations).

Telangiectases do not call for any therapeutic or surgical treatment; however, angiomas should be given efficient treatment as early as possible, as in old age they may turn into malignant tumours.

Radium therapy is currently considered one of the best treatments for angiomas. Less effective is surgical treatment: cauterisation by carbon dioxide snow and alcohol injections into the depth of the angioma.

The most frequently observed deformities of the *alimentary tract* are "harelip"—a fissure of the upper lip due to failure of fusion of embryonic facial processes, and cleft palate—a fissure in the hard palate due to failure of fusion of both halves of the hard palate. These deformities of the mouth call for surgery that is performed at two or three months for harelip, and not before five years for cleft palate (so that the fissure be closed by the time the child starts school).

Besides the above anomalies and deformities a great number of deviations from normal are observed in children: six digits on hands and feet; fusion of digits; partial or complete absence of digits on hands and feet; imperforate anus (due to the opening being closed by a membrane of skin, or to defective development of the rectum); congenitally turned-in toes; hypospadias—the urethra does not open in the centre of the penis, as it should, but lower, usually at the site of the frenum; congenital dropsy of the brain (hydrocephaly) due to its defective development, or to in-born syphilis; microcephaly—a small head due to defective development of the brain; hernia of the brain protruding over the bridge of the nose or at the back of the head; obstruction or atresia of the bile ducts; constriction of the pyloric opening of the stomach; dilation of the large intestine; spina bifida—a defect in the closure of the spinal canal.

Transitory (Dehydration) Fever

Transitory fever of the newborn appears on the second to sixth day of life, more frequently in heavy infants; it is accompanied by weight loss and paleness. The disease is the result of dehydration (fever caused by thirst), and it is quite frequently observed when the mother's milk is late in coming, when breast nursing is delayed for too long, and in weak sucking. The infant's temperature rises to 38-39°C. Besides the newborn, this disease is also observed in infants of a more advanced age as a result of inadequate fluid intake. The fever is rarely encountered when infants are put to the breast six to twelve hours after birth and take in enough fluids.

Rapid recovery is evoked by giving water or Ringer solution with glucose (Sol. Ringeri 100.0, Glucosae 5.0. DS. one teaspoonful 4-6 times a day).

Sexual Crisis in the Newborn

Engorgement of the breast is often observed in newborn infants of both sexes; the dimensions of the gland may attain that of a large nut. Colostrum may be secreted or pressed out of the engorged breast. However, the manual expression of this "milk" that is sometimes performed should never be practised, as this may injure the tissue of the gland, infect it with pyogenic microbes and lead to a suppurative inflammation (mastitis of the newborn), resulting in a subsequent disruption of the normal function of the mammary gland when the girl baby becomes a woman.

Scrotal edema is often observed in male infants, and a *swelling and redness of the major and minor lips (labia majus and minus) and of the clitoris* in females. At times a mucous discharge from the vagina may be present—*vulvovaginitis of the newborn*.

A much rarer occurrence is the appearance of a bloody discharge from the vaginal orifice, and even red blood similar to menstruation; this discharge may appear within the first 3-4 days after birth and last two to five days, with no relapse. Such phenomena are now considered to be a reaction of the newborn infant to the maternal hormones transmitted to the fetus through the circulating blood, and retained in the system postnatally.

Jaundice in the Newborn

Yellowness of the skin is observed in varying degrees in the majority of newborn infants delivered at term, and in all premature infants (over 90 per cent). This is so-called *simple or physiological jaundice*. Usually the entire surface of the skin is affected, the oral mucosa slightly, and the sclera rarely. The colour intensifies during the first 3-4 days, to disappear gradually by the 7th-10th day; however, at times physiological jaundice may persist for a whole month. The soles and palms are usually unaffected, no bile pigments are found in the urine, stools are not discoloured, but the blood bilirubin level is elevated. This type of jaundice in the newborn is due to the inability of the immature liver to deal with the increased load of blood pigment (bilirubin) resulting from rapid hemolysis of superfluous red cells. Another cause of jaundice may be that in some newborn the biliary pigments are still delivered into circulation at the moment of birth and during the following 3-4 days as in prenatal life, owing to the incomplete dilation of the common bile duct (*ductus choledochus*).

In some cases physiological jaundice of the newborn may be due to both causes, one or the other prevailing.

In rare cases other types of jaundice are observed in the newborn.

Jaundice due to congenital immaturity of the bile ducts is manifested by a dark yellow colouration of the skin and sclera, discolouration of the stool, dark urine; the liver is always enlarged in these pathological cases. This is a developmental anomaly that can only be dealt with surgically, and even then only in cases of obstruction or atresia of the common bile duct, provided the gall-bladder exists. The operation consists of suturing the gallbladder to the duodenum.

Jaundice of the newborn may be caused by sepsis—*septic jaundice*; by syphilis—*syphilitic jaundice*. Rarely jaundice may be the manifestation of epidemic hepatitis (Botkin's disease).

Finally, the newborn may be afflicted by a severe type of jaundice that is the result of lesions of certain areas of the brain and its basal ganglions; this type of jaundice is frequently accompanied by convulsions and various subsequent nervous phenomena (pareses, paralyses). Postmortem examinations show biliary pigmentation of these areas of the brain. This disease is called *nuclear jaundice* (kernicterus) and is a form of hemolytic disease in the newborn, sometimes observed in several offspring of the same parents. It was discovered not so long ago that this disease is due to hemolysis of the infant's blood caused by the absence in the mother's blood of the so-called rhesus-factor, Rh. Mortality in this type of jaundice is very high, and even if the child survives he may be affected by pareses and paralyses. Another form of hemolytic disease is severe progressive anemia of the newborn without jaundice.

Asphyxia of the Newborn

Asphyxia is not essentially a disease, it is only a symptom evoked by a number of causes. Thus there is *congenital* or *intra-uterine asphyxia* that develops within the uterus owing to oxygen deficiencies during delivery, and *acquired asphyxia* that develops postnately owing to complete or partial respiratory incompetence.

Oxygen deficiency in the fetus during delivery may be caused by a number of disturbances of placental circulation, such as premature detachment of the placenta (the afterbirth), a tightly twisted or compressed cord, and so forth.

Congenital asphyxia may be mild, the so-called blue asphyxia (asphyxia livida), or of a more severe form—asphyxia pallida.

Blue asphyxia is characterised by a cyanotic colouration of the skin, rare respirations, decelerated heartbeats attended by satisfactory muscle tone and normal or slightly lowered response to stimuli.

Asphyxia pallida is attended by a very pale skin and extremely cyanotic lips; the pulse is slow and feeble, but in far-gone cases it may be very rapid. Muscular tone and all reflexes are lowered.

In blue asphyxia the intervals between respirations are highly prolonged, but the child does breathe; *asphyxia pallida* is accompanied by complete absence of respiration.

Acquired asphyxia is characterised by cyanosis; it depends on certain congenital heart defects, atelectatic lungs, inborn lesions of the central nervous system, particularly of the respiratory centre, as a result of brain hemorrhages. In such cases the fetus does not experience oxygen deficiency during the act of birth, and it is often born without any signs of asphyxia; however, cyanosis follows soon after the cord is cut.

Prognosis is grave, particularly in congenital *asphyxia pallida*.

Treatment. The mucus is withdrawn from the throat and mouth by suction through a soft catheter attached to a rubber syringe; alternate cold (15°C) and hot (39°C) baths, artificial respiration, oxygen, subcutaneous injections of caffeine, lobeline and camphor oil are given. Close observance of such infants is necessary, as relapses of asphyxia are possible.

Scleredema and Sclerema

Scleredema is edema of the subcutaneous (subdermal) tissues in asthenic and premature infants attended by weakened cardiac activity and low temperature. The edema appears on the skin of the legs, but may also affect the arms; it pits upon pressure; the skin is pale and shiny.

Sclerema is a hardening of the subcutaneous fat tissue. It is manifested by the appearance on the legs and feet of a hard pale edema that does not pit upon pressure. Sclerema affects premature infants and accompanies grave disorders of nutrition; prognosis is bad.

Treatment. Hot baths, hot-water bottles, caffeine internally and subcutaneously.

Diseases Due to Birth Injuries

Sometimes the newborn infant is gravely affected by the birth act, and he sustains a number of injuries.

Birth tumour of the head (caput succedaneum). This is a limited

swelling of the skin caused by pressure either in the uterus, or while the head is passing through the narrow lower part of the pelvis. Its formation is due to serous infiltration of the subcutaneous connective tissue. This swelling disappears sooner or later, depending on its dimensions. In children of multiparas (women who have borne more than one child) it disappears within several hours, while in first-born infants it is sometimes noticeable for days.

Hemorrhages. Hemorrhages may be caused by injuries sustained during delivery; they depend on the brittleness of the blood vessels, on the difference between atmospheric and intrauterine pressure.

Hemorrhages in the skin occur in dimensions ranging from petechia (minute spots of hemorrhage) to quite noticeable black marks. Usually all skin hemorrhages clear rapidly if there are no abrasions nor injuries of the skin; deeper lesions caused by forceps blades during delivery may serve as an entrance point for various infections; to avoid this abrasions of the skin should be painted with iodine directly after birth, while deeper lesions call for sterile dressing.

Hemorrhages into muscles (hematomas) occur mostly on the neck, in the sternocleidomastoid muscles; they may subsequently result in wryneck (torticollis). Hematomas of the neck muscles usually disappear spontaneously within several weeks. If a tendency to wryneck is noticed in the second week of life warm baths are recommended (38-39°C) and light massage of the affected side with passive turns of the head. In instances of persistent wryneck accompanied by a sharp contraction of the sternocleidomastoid line surgery has to be resorted to subsequently.

Hemorrhagic tumour of the head (cephalohematoma) is caused by an accumulation of blood from ruptured capillaries between the periosteum and a skull bone in the parietal region. Its occurrence is commonly on one side of the head; a firm ring can be palpated at the periphery of the hematoma. The hematoma takes quite a while to resolve and hardens significantly owing to the deposit of calcium. Infectious infiltration may cause suppuration (Fig. 18).



Fig. 18. Cephalohematoma on right parietal bone

Treatment. A soft bandage to avoid bruises and infection; if an abscess forms it should be incised.

Intracranial hemorrhages (subdural or subarachnoid) may be caused by birth traumas, cracks, fractures and depressed fractures of the skull bones. Ruptures of the sinuses are fatal. Intracranial hemorrhages often evoke convulsions, somnolence, at times hemiplegia, ptosis, paresis of the facial nerve. The spinal fluid elicited by lumbar puncture often shows blood and does not clear up by centrifugation (hemolysis). Intracranial hemorrhage may also be due to a vitamin K deficiency.

Prognosis is grave, the mortality rate is a high one.

Treatment is symptomatic: ice to the head, a chloral hydrate enema, luminal for convulsions, rest, hemotherapy (10 ml intramuscularly), careful feeding, elevated head, calcium chloride.

Internal hemorrhages are discovered on postmortem examination. Hemorrhage into the adrenals is a cause of rapid death. Ruptures of the liver and spleen also give rise to internal hemorrhages and their termination is fatal.

Injuries and fractures of bones. During delivery, particularly if forceps are applied, various bones of the body may be injured or fractured; however, the following traumas are encountered most commonly.

Depressed fractures of the skull occur chiefly in the anterior part of the parietal bones, less often in the area of the parietal eminence (tuber parietale). Depression of the skull bones does not usually evoke any symptoms of brain pressure and calls for no treatment; only in cases of very deep depressions do symptoms of brain disturbance (convulsions, paralyses) appear immediately after birth. Such cases call for immediate surgery.

Cracks in skull bones mostly appear together with depressions, but may occur independently. They are usually localised in the areas of the parietal and frontal bones; they produce no symptoms and call for no treatment.

Fractures of clavicle are more frequent in deliveries with obstetric aids, although they do occur during spontaneous deliveries too; the course of these fractures is benign, healing is good and does not call for any treatment in most cases. A complete fracture may necessitate a light bandage for immobilisation of arm and shoulder; in 7-8 days complete healing occurs, with good callus formation. The callus resolves subsequently.

Fractures of humerus are comparatively uncommon, but when they do occur the fragments often become displaced and there-

fore a special immobilising bandage has to be applied and greater precautions taken.

Fractures of femur are also not frequent occurrences; they call for traction and a plaster cast.

Diseases of the Umbilicus

The umbilicus includes two umbilical arteries, the umbilical vein, the rudimentary allantois and residual omphalomesenteric duct embedded in embryonal tissue (Wharton's jelly). After the cord has been cut the navel should not be dressed frequently—only when the dressing becomes soiled. The remnant of the cord sloughs on the 7th to 10th day of life; iodine may be applied to the wound, but the usual procedure at present is the application of a 2 or 5 per cent solution of silver nitrate (after first washing out the wound with hydrogen peroxide) or in more persistent cases, a dermatol ointment (bismuth subgallate) dressing, or pure dermatol powder. If the wound hemorrhages it should be cauterised with 5 per cent silver nitrate; persistent hemorrhages are treated with subcutaneous injections of diphtherial serum (10 ml). Slight exudation from the navel after the cord has sloughed is treated with daily applications of 70° alcohol. An ulcer in the ground of the navel should be dressed daily with a 2 per cent solution of silver nitrate (after washing the wound with hydrogen peroxide).

Infections of the umbilicus. Prior to sloughing of the cord contaminated amniotic fluid may be a source of infection.

After the cord has fallen off the navel wound may become infected through nonsterile dressings, hands of the staff, and bath water. That is why it is not advisable to bathe newborn infants before the cord has sloughed.

Slight umbilical infections may be accompanied only by retarded healing of the navel and a constant exudation of a serous, slightly turbid fluid ("wet navel"). The constant irritation due to a wet navel may lead to the growth of a granuloma and the formation of a so-called fungus of the navel (*fungus umbilici*), an elevation over the bottom of the umbilical depression the size of a pea. If these granulations are connected to the body by a small stem a thin thread of sterile silk is tied around this stem and the granulation sloughs off in a short time. In cases where the granulations fill up the entire bed of the navel depression they are cauterised with a silver nitrate stick, care being taken not to touch the edges of surrounding skin.

More severe infections of the umbilicus may lead to the formation of slow-healing suppurative *navel ulcers* accompanied by inflammation of the skin surrounding the umbilical wound, *omphalitis*, while the spreading of the process into the subdermal tissues leads to the formation of an umbilical *phlegmon*. In weak and emaciated children necrosis of the affected area—*umbilical gangrene*—may occur.

The spreading of the suppurative process from the umbilicus and umbilical cicatrix to the umbilical vessels may evoke general sepsis in the first days of life.

Penetration of diphtheria bacilli into the navel may result in *umbilical diphtheria*, while *tetanus* may be induced by infection with the tetanus bacillus.

Prophylaxis and treatment of diseases of the umbilicus. Any dressing of the umbilical stump calls for scrupulous cleanliness of hands and dressing material. For some time after the cord has sloughed off the child should be bathed only in boiled water.

Sterile dressing is used for umbilical diseases; phlegmons should be lanced and the child given penicillin therapy.

Umbilical hernia is often encountered in infancy; this condition is the result of a weak development of the muscular ring and subdermal tissue around the navel, and is mostly observed in feeble and premature infants. Extension of the abdomen, hard crying and coughing cause the peritoneum and skin to protrude, forming a hernial sack containing intestinal loops. Hernias are usually easily reduced. Strangulation is rare. Real umbilical hernia should be distinguished from the so-called "skin navel" condition that may form when the umbilical ring is closed properly. "Skin navel" is due to a superfluous piece of skin in the area of the navel ring. Umbilical hernia usually clears spontaneously by 12 to 24 months, when the child has become stronger, and surgery is rarely required.

In order to accelerate the disappearance of umbilical hernia it is recommended to place the child on his stomach for some time every day. Adhesive tape, particularly in the first 2-3 weeks of life, is not recommended as the tender skin around the navel is easily irritated and becomes moist.

Sepsis of the Newborn

Sepsis is a generalised disease of the organism evoked by constant or intermittent penetration of microbes into the blood current from a known or obscure seat of infection as a result of the weakening of the protective apparatus (immunity). In the blood

of adults, with their well-developed protective systems, the microbes soon perish and sepsis occurs much less frequently. The general low resistance of infants, particularly of the newborn, is the cause of frequently observed septic processes. The source of sepsis in the newborn is most commonly the umbilicus, then scratches (abrasions), irritation and pustules on the skin, abrasions of the oral mucosa due to bad management in wiping out the mouth, nasopharyngites during which the infection spreads to the ears and periantral (mastoid process) cells, then to the intestine, and, finally, pulmonary processes involving the pleura, pericardium, peritoneum, the dura mater and the joints.

The septic pathogens are mostly streptococci and staphylococci, less frequently diplococci, the influenza bacillus, and other microbes that sometimes show a growth in cultures of blood specimens taken from the living body, and are commonly found in suppurative sites during autopsies. Sources of infection are contaminated waters and lochia, soiled bedclothes and objects used in the care of patients, insufficiently clean hands of the midwife in the delivery room, dirty hands of the nursery staff, as well as the affection of the staff or the mother by pyoderma or paronychia. The development of intrauterine sepsis is very rare. Septic postparturition diseases in the mother do not always result in sepsis in the child. Child-birth at home is frequently a cause of sepsis. Prematurity and feebleness, syphilis and severe birth injuries make infants particularly sensitive to sepsis.

Pathologic anatomy. Postmortem findings in cases of umbilical sepsis usually show purulent thrombi in the umbilical vessels; these thrombi may be located at some distance from the umbilical wound that has already formed a good cicatrix. Moreover, phlegmons, peritonitis, suppurative pneumonias and metastases in the joints are not infrequent. In cases of other sources of sepsis the primary seat of suppuration may be discovered in various organs (the skin, viscera, bones).

Clinical findings in sepsis are sufficiently specific to diagnose most cases properly, these diagnoses being confirmed by post-mortem examinations. The early signs of sepsis of various origin are the following:

Loss of appetite, refusal of breast, poor nursing. However, these symptoms may be observed in nonseptic premature and feeble infants, and in cases where the mother's milk does not flow freely (tight breast).

Rapid loss of weight. Sometimes the infant loses as much as 300-600 g, or even 1 kg by the time he is dismissed from the ma-

ternity hospital. But babies also lose weight when they fail to nurse vigorously enough and when the mother's milk supply is insufficient, and these factors must be taken into account when making the diagnosis. Frequent loose bowel movements, at times orange-coloured; vomiting is rare.

Greyish skin instead of the usual pink, often with a touch of jaundice and cyanosis. Sometimes a measles-like or hemorrhagic rash appears on the skin. Later sclerema develops. The lips and nails are usually cyanotic.

Dulled heart sounds and accelerated pulse.

In three-fourths of the cases the temperature is elevated, mostly subfebrile. At times it may rise for a day to 39-40°.

Upon recovery the temperature may stay at 37.1-37.3° for prolonged periods, although the general health of the child is good and he gains in weight (latent infection). Finally, a temporary period of normal temperature may be followed by a relapse of fever. In one-quarter of sepsis cases the temperature is normal throughout the process, and this should always be borne in mind (obscure sepsis).

The spleen is usually neither enlarged nor palpable, but the liver is always significantly enlarged. In some cases of umbilical sepsis tension of the rectus abdominus muscle (the Krasnobayev symptom) is apparent. When the process involves the peritoneum, the abdomen becomes hard and painful. However, subsequently, prior to death and attendant on peritonitis, the abdomen sometimes becomes soft. Finally, in certain cases edema of the abdominal wall is observed. In half of the cases of umbilical sepsis the sloughing of the cord is delayed. In the area of the navel a local scab is formed, or a purulent or hemorrhagic-suppurative discharge is observed, sometimes a swelling and redness of the skin surrounding the navel; on the contrary, in a number of cases the umbilical stump is absolutely normal externally and no exudate is present. Moreover, a purulent exudate from the navel may accompany a simple case of omphalitis, not being a symptom of umbilical sepsis alone.

Urinalysis often shows albumin, red blood cells, less frequently hyaline and granular casts. Leukocytosis and neutrophilia may be observed in the blood, as well as monocytosis and a slight acceleration of the ESR. Eventually, significant anemia may develop.

In the most severe cases hemorrhagic vomiting and black feces (septic melena) occur infrequently. Stupor and convulsions are frequently seen. Occasionally pyogenic meningitides set in. The general condition is usually grave.

In *dermal sepsis* the process often begins with a scratch, diaper rash or small pustule; owing to the immaturity of the dermal barrier these lesions rapidly turn into phlegmons. The above-mentioned symptoms of sepsis appear soon. Phlegmons of the back and neck are the most severe. However, not every phlegmon in the newborn should be looked upon as dermal sepsis; many phlegmons clear up under proper general and surgical treatment.

Oral sepsis is accompanied by a swelling of the cervical glands and the usual symptoms of sepsis.

A peculiar form of sepsis is the so-called *toxico-septic diseases* of the newborn. The clinical findings are as follows: at the beginning, on the 9th to 12th day of life, the infant becomes pale, flabby, listless at the breast; later vomiting sets in and the stools become very loose and green, soon taking on a vivid orange colour owing to an abundance of bilirubin. The temperature elevates to 38-39°. Often an intensive shortness of breath appears, the child's nostrils blowing out with every respiration, while percussion and auscultation of the lungs at first yield nothing. X-rays show a sharp distension of the lungs, and subsequently the formation of small nidi in the pulmonary tissue. Upon auscultation some moist and dry crepitations are noted. Postmortem examinations also show a limited number of small nidi that upon microscopic examination prove to be interstitial pneumonia. Infection is evidently transmitted through contaminated lochia, soiled bed-clothes and objects used in the care of mother and child, dishes and utensils. *Escherichia coli* is found frequently in the feces, occasionally staphylococci. This form of disease is frequently of a grave nature and its mortality rate is high. The infection probably penetrates through the mouth; postmortem examinations show no changes in the umbilical vessels.

All types of sepsis are often aggravated by pneumonia, parenteral dyspepsia, less frequently by otitis and pyelitis.

Diagnosis of sepsis is determined on the grounds of the above symptoms. However, it is not in every case that all the symptoms are present. It is often possible to detect the nidus of infection (the navel, the skin), but its source remains obscure.

Prognosis for sepsis is very grave. The later the diagnosis has been made and treatment begun, the higher the mortality rate.

Prophylaxis. A number of prophylactic measures may be recommended, such as smaller nursery wards (no more than 15-20 babies to a ward), and division of wards into individual cubicles; sufficient warmth (20°C) and humidity (60-70 per cent) in the premises; good ventilation of premises and irradiation with ultraviolet

ray lamps (sun lamps); a sufficient number of trained staff in the nurseries (no more than 15-20 babies under the care of each nurse); isolation of babies from sick mothers; a sufficient supply of diapers (25 per baby) and shirts or kimonas; thorough washing and sterilisation of all body and bed-clothing (or ironing on both sides after washing); scrupulously clean premises, bodies and hands of medical staff and mothers; face-gags for nursery staff and mothers in cases of infectious diseases; timely dismissal of pyoderma-affected personnel.

The umbilical stump should be dressed, both in the hospital and at home, by a doctor or nurse upon definite indications only (odour, discharge, red skin around the navel). In these cases the navel is washed out with hydrogen peroxide and a 2 per cent solution of silver nitrate is applied to it, or a solution of penicillin; dermatol (bismuth subgallate) ointment or powder are also used. The dressing is changed regularly. The best dressing is an ordinary sterile gauze belt passed around the body over the navel. Adhesive dressings should be avoided.

The mouth should be wiped out carefully, and only by doctor's orders, to avoid hurting the mucosa. In recurring cases of sepsis the maternity hospital is closed down and then thoroughly disinfected and all the rooms are treated by ultraviolet radiation.

Treatment. An affected baby must be isolated or hospitalised immediately. He must be provided with good care and a sufficient supply of breast milk directly from the breast, or by bottle, spoon, dropper or a tube. Vigorous treatment should be given from the very beginning in the maternity hospital. Sufficient warmth (20°C) is essential, as well as humidity (60-70°), also fresh air and light in the ward. Penicillin injections are given immediately intramuscularly (in the outer aspect of the thigh) 2-3 times a day for seven to twelve days. In the most severe cases penicillin is injected once into a vein of the head and three times intramuscularly. The total daily penicillin dose per kg of body weight is 10,000-15,000 u given in two or three injections.

Penicillin is administered together with sulfonamides (sulfa drugs), 0.2 g per kg of weight daily, in four doses, for six to twelve days. In weight losses subcutaneous injections of 50 ml of sterile 5 per cent glucose are indicated. For improving general tonicity transfusions of citrate blood are given over intervals of 3-5 days into the veins of the head, 5 to 10 ml per kg of weight, or measles convalescent serum, 10 to 20 ml intramuscularly for 10 days, or maternal blood, 5-10 ml on alternate days into the muscles of the thigh. Cardiac preparations are also necessary (a 10 per cent

solution of caffeine, 0.3 g of camphor injected subcutaneously three times a day).

If any discharge from the navel is noticed the umbilical wound is washed out with hydrogen peroxide, and then a 5 per cent solution of silver nitrate is applied; a compress may also be beneficial (a 1:1,000 solution of rivanol or sterile physiological salt solution). All the suppurative seats of infection must be lanced in time. Pus is withdrawn from the joints with a 5-gram syringe and 10,000 u of penicillin are injected into the cavity of the joint.

Timely lancing of the phlegmons in dermal sepsis is important. Mastoiditis often heals without surgical treatment when early complex therapy is applied together with super-high frequency radiation (10 to 15 treatments); if no effect is obtained by these methods mastoidectomy (antrotomy) is obligatory. Penicillin often proves effective in the treatment of abscessing toxic pneumonia. If stools of septic patients become orange-coloured penicillin should be given together with albuclid (dose as other sulfa drugs).

Tetanus in the Newborn

Tetanus is transmitted through the umbilical wound when proper hygiene has not been observed in the care of the newborn. This happens only in cases when the child is delivered without appropriate obstetric aid (not in the hospital, but on the street or in the field). The initial sign of the disease is *lockjaw* (trismus, Fig. 19).



Fig. 19. Tetany in a newborn

The mother notices that the baby closes his jaws convulsively on the nipple when he is put to the breast. A general pattern of convulsions and reflex attacks is then formed.

Often parenteral dyspepsia and malnutrition develop. The temperature is frequently elevated. Tetanus of the newborn is very dangerous; it has a rapid, fatal termination (3-5 days) and is attended by pneumonia, severe convulsions and cardiac disorders.

The mortality rate among the newborn is 80-100 per cent.

Serum therapy. The difficulty of this method of treatment is that by the time the clinical symptoms of the disease become evident, the toxin has already deeply affected the central nervous system and the therapeutic effect of the serum is not very reliable. A combined method of treatment is employed—simultaneous intramuscular and spinal injection of the serum.

Difficulties are encountered in injecting the serum into the vertebral canal owing to intensive opisthotonus and high reflex excitation. Therefore, prior to injections the infant should be given an enema containing 0.5-1.0 g of chloral hydrate. 10,000 antitoxin units are injected into the spinal cord and then 50,000-100,000 u intramuscularly daily for three to five days. This treatment is repeated every day, or lumbar injections are alternated with intramuscular injections until a significant alleviation of the symptoms has been attained.

Symptomatic therapy. Preparations lowering the excitability of the central nervous system are used: chloral hydrate in enemas (a 2 per cent solution, 25-30 ml for infants, 50 ml for older children) 3-4 times a day; a 2 per cent solution of sodium bromide is given orally.

Care, diet, treatment. The basic rule of treatment is *absolute rest and silence*. It is desirable to isolate the patient in a darkened room. The staff must maintain complete silence. Handling of the patient must be minimised and done as carefully as possible; no loud talking is allowed in his presence, as even the slightest stimulus may evoke convulsions.

The patient is given liquid and semiliquid food, often from a spoon or a small teapot, as he cannot open his mouth properly. A sufficient supply of liquid (sweet tea, glucose solution) is important. In cases of difficulty in swallowing the child is given physiological saline solution enemas with 5 per cent solutions of glucose, or Ringer solution and glucose (5 per cent) are injected subcutaneously (100-500 ml). Infants are given breast milk through a tube if necessary. One of the best sedatives is a hot bath (38-40°C) once or twice a day for 10-12 minutes.

Prophylaxis. The most cautious aseptic care should be taken of the umbilical wound of the newborn.

In cases of street deliveries, when the infant's skin may have been contaminated by earth and dirt, a *prophylactic injection* is necessary (10,000 u of tetanus antitoxin).

Premature Infants

A premature infant is one that is born earlier than 280 days after conception. Premature infants are born more often to primiparas than to multiparas.

It is often difficult to determine the cause of prematurity. The most frequent causes are various diseases of the mother: syphilis, tuberculosis, malaria, chronic nephritis, chronic diseases of the liver, acute severe infections (scarlet fever, typhoid). Moreover, premature deliveries may be caused by repeated artificial abortions and by various diseases of the woman's reproductive organs.

Accidents (falls and blows on the stomach), lifting heavy weights, hard physical work may also cause premature deliveries.

Premature births may likewise be the result of malnutrition of the expectant mother and vitamin deficiencies. Child-birth may also be brought on before term by mental shock, as well as by serious disorders of the mother's nervous system.

Symptoms of prematurity in the infant: weight less than 2,500 g; crown-heel length less than 45 cm; head unproportionally large in comparison with the trunk; besides the anterior fontanel the posterior and lateral fontanels are also open; the ears are very soft and adhere closely to the head; the skin is dry, wrinkled and covered with down (lanugo); the nails do not always cover the entire nail bed; in boy infants the testicles are undescended; in girls the labia minus (the minor genital lips) protrude from the labia majus (the major genital lips); the infant's voice is only a feeble chirp.

The functions of the nervous system in premature infants are still more erratic than in full-term babies. Therefore, respiration is irregular and superficial. Premature infants often suffer attacks of cyanosis owing to the immature function of their respiratory tract. Moreover, significant atelectasis is often observed (imperfect expansion of the lungs), so that the respiratory surface of the lungs is greatly decreased. *Immature heat-regulation* leads to a rapid lowering of the bodily temperature to 35°, or to a rapid overheating to 40°. Finally, *premature infants often do not take the breast, nor can they suck from a bottle or be fed from a spoon*

or a medicine dropper owing to their weak sucking and swallowing reflexes.

The *immunity* of premature infants is insufficient for the same reasons, and their ability to handle infections is therefore decreased (e.g., influenza and pneumonia).

The *vascular system* of the premature is very delicate; skin and brain hemorrhages are often caused by delivery. Moreover, subsequently these children very frequently suffer from *rickets* and *anemia*.

Provided that premature infants are properly managed and fed their rate of development is much higher than that of full-term children. If the latter triples his weight by 12 months, then the premature infant increases his weight fivefold, and in certain cases (very low weight at birth) even sixfold and more.

Often a healthy premature infant weighing no less than 2,000 g at birth catches up by one year (in weight) with a full-term baby of the same age.

Foreign authors cite a high mortality rate among the premature, attaining 45 per cent for babies weighing 1,500-2,000 g at birth, and 30 per cent for those weighing 2,000-2,500 g. These figures have now been greatly lowered in the Soviet Union. In certain institutions (Premature Department of the U.S.S.R. Academy of Medical Sciences Institute for Pediatrics) mortality among 81.5 per cent of premature infants born weighing less than 2,000 g was brought down to zero.

It has been demonstrated by observations made in recent years that the subsequent development of premature babies follows a normal course both physiologically and mentally, and they grow up nowise inferior to full-term individuals of the same age.

Care of the premature. Formerly premature babies were placed in closed glass cases—*couveuses*. Now this type of incubator is not used at all. Every maternity hospital has a special ward for premature infants, where particular care is taken of these babies. Directly after delivery the infant is swabbed with warm vegetable or mineral oil and dressed in warm stockings, a vest made of quilted gauze and cotton wool, and a cotton-wool cap. Three hot-water bottles or rubber bags are placed at the infant's feet and sides (the temperature of the water is about 45°C). If the baby becomes overheated (elevation of body temperature to 38-39°) the heating devices are removed. When changing the baby the diapers should be warm, the entire procedure completed as swiftly as possible. The bath of the premature infant should be no cooler than 38-39°C, and the room temperature not lower than 20°C. The

bath should be given as rapidly as possible, and the child wrapped in warm blankets or diapers after his bath.

The first bath is given after the cord has sloughed off, and then every 2-3 days. Before making the baby's toilet the mother's or nurse's hands should be thoroughly washed and then warmed. Persons with pyogenic eruptions on their hands, or with the grippe, must not be allowed to care for babies. Premature infants very frequently become asphyxiated, and they succumb to these attacks if proper measures are not taken urgently. In these cases the child is given 1-2 oxygen bags, and during attacks also 0.2-0.3 ml of a 10 per cent solution of caffeine subcutaneously, or a 0.5 per cent solution internally, and also respiration stimulants (lobeline, cytitone).

If the *baby takes the breast* he is nursed every two hours, ten times in 24 hours, with a six-hour break at night. Sucking usually fatigues the premature easily, therefore they are nursed at the breast from two to five times; the other five feedings are expressed breast milk given in adequate quantities. If the infant fails to withdraw enough milk from the breast he is given an additional portion of breast milk in a bottle. When the baby does not take the breast at all he should be fed ten times on expressed breast milk. If he cannot suck from a bottle he is fed with a spoon, or with a medicine dropper through the nose. In cases when the infant cannot swallow at all he must be fed through a catheter (size 12-15) 6-7 times a day. As the infant grows, he is gradually trained to the bottle, and then put to the breast.

Premature infants require more calories than ordinary babies do—140 to 150 cal/kg. If the mother produces too little milk the best supplement is manually expressed milk obtained from another woman, or acidified buttermilk with 5 per cent sugar added. Ordinary formulas with cereal water are sometimes also satisfactory when given as a supplement. If no breast milk at all is available the premature infant may be given the usual milk-cereal water formulas and 1-2 bottles of sweetened buttermilk a day. Low weight gains necessitate a higher protein content in the food; 2-4 per cent of repeatedly sieved curds (one half to one teaspoonful) or one-fourth teaspoonful of the protein preparation plasmon are added to a bottle of formula and the entire mixture is brought to a boil; before feeding the mixture should be shaken thoroughly. As the premature are often afflicted by rickets they must be given, beginning at the age of two months, fruit juices and fish-liver oil, starting with 2-3 drops and ending with one half to one teaspoonful twice a day. Outdoor walks in the cold season are permitted only after the child weighs 3 kg.

DISTURBANCES OF DIGESTION AND NUTRITION IN INFANTS

The term digestive disturbances signifies generalised ailments that are not manifested by gastrointestinal symptoms alone (diarrhea, regurgitation, vomiting), but also by general disturbances of metabolism and of the activity of all organs and tissues. Nutritional disorders are generalised disturbances accompanied by weight and growth lags, often with no gastrointestinal symptoms (diarrhea or vomiting). Every digestive disorder evokes a more or less significant nutritional disturbance. Lastly, nutritional disturbances often lead to digestive trouble that is much more severe than when healthy children are affected. That is why we now speak of "disturbances of digestion and nutrition" and do not divide these usually concomitant phenomena.

Disturbances of digestion and nutrition are more commonly observed in infancy. They take first place in infant mortality (up to 30 per cent of all deaths in the first year of life). Breast-fed babies are affected much less and in much milder forms than bottle-fed infants. The mortality rate among the latter is eight times higher.

The entire aspect of the child changes in digestive and nutritional diseases, his weight and growth are retarded. The weight curve becomes flatter than normal, weight gains stop, and finally more or less rapid weight losses are observed, the child becoming very thin. The difference between morning and evening temperature becomes more noticeable (0.5° and more), there are sharp rises and falls in the temperature; the turgor of the skin decreases and it becomes palish-grey. Owing to emaciation and the rapid decrease of subdermal fat wrinkles appear on the forehead and creases on the thighs; the muscles become either hypotonic or hypertonic. Sleep is disturbed, the child cries frequently, at times is drowsy or develops convulsions. Pulse and respiration become irregular; the stools are loose and frequent. Static functions are retarded as compared with healthy babies of the same age (slow to sit and to walk), and so are the mental capacities (the baby does not talk, smiles rarely).

The factors that play an essential part in the deterioration of the general condition of the child and of his digestion and nutrition are poor care, stuffy air, tight clothing, overheating, rooms not aired, not enough time outdoors, soiled clothing and objects used in the management and care of the child (dishes, pacifiers, nipples, bottles, utensils), dirty hands of the persons caring for

the child, flies, insufficient drinking water between meals in the summer, erratic feeding (irregular feeding every time the baby cries both in the daytime and at night, overfeeding with breast milk, too early addition of solids, overfeeding with too much cow's milk or fat mixtures, particularly in the summer), undernourishment when the mother does not produce enough milk, highly diluted cow's milk (two-thirds water), an insufficient daily quantity of milk formulas, underfeeding.

Cow's milk constituents most important in disturbing digestion are carbohydrates and fats. Fermenting under the action of bacteria, carbohydrates irritate the intestinal wall, leading to a decrease in the secretion of enzymes and intestinal juices; this evokes intensive peristalsis, extension of the abdomen due to the formation of gas, diarrhea, regurgitation and vomiting, i.e., a digestive disorder. Lactic acid has the greatest tendency to fermentation. An increase in carbohydrates is often the cause of digestive and nutritional disturbances. A carbohydrate deficiency may also lead to nutritional disorders. If the child is overfed with fat mixtures the fats are decomposed by bacteria into the lower fatty acids; the latter irritate the intestinal walls and may also lead to digestive trouble. Prolonged overfeeding with fat mixtures leads to complete nonassimilation of milk and to the appearance of a saponaceous fatty stool (decomposition and the binding of the lower fatty acids with calcium results in the formation of fatty soaps that make up the bulk of the saponaceous fatty feces). Prolonged elimination of fats from the child's diet lowers his general condition and his resistance to infections. Food proteins inhibit carbohydrate fermentation and they are therefore an important therapeutic factor in the treatment of digestive and nutritional disorders. The salt content is important for the retention of water in the body together with carbohydrates; in combination with other food constituents the salt level evidently plays some part in the etiology of nutritional disorders. If a child is not given water between meals in the summer excessive perspiration and evaporation of water may lead to "thirst fever" (elevation of temperature up to 38-39°) and subsequent digestive trouble.

Intensive loss of water (dehydration) during toxic dyspepsia is concomitant with a grave general condition. Vitamin deficiencies may also play a certain part in the etiology of nutritional and digestive disorders. Excessive amounts of fruit juice often cause diarrhea owing to the irritation produced by the organic acids contained in these juices.

An important part in the etiology of nutritional and digestive diseases is played by bacterial infections in the intestine. The infection may penetrate through the mouth (exogenous infections); sometimes the bacteria that live in the lower part of the intestine infect the higher sections that are usually free of bacteria.

In the summer milk is infected by various microbes more easily; the microbes multiply rapidly and form poisonous toxins (exogenous infection). Unboiled, sour, or impure milk may evoke severe digestive disorders. Boiling kills the microbes, only their spores remain; these spores cannot develop at low temperatures, therefore boiled milk kept cold is much less dangerous than raw milk. However, bad milk that has been partly decomposed by the action of bacteria may still cause severe digestive disorders even after prolonged boiling, as many products of bacterial activity are not destroyed by boiling. I. Mechnikov and P. Tsiklinskaya considered that there exist specific pathogens of toxic dyspepsia, namely *B. proteus vulgaris*. There is a group of specific *dysenteric bacilli* that are the cause of an epidemic disease among children in the summer—dysentery. Rare digestive pathogens are the *paratyphoid bacilli* (types A and B). Finally, there is a so-called *parenteral infection* that also impairs digestion. In the course of many pyrexial illnesses (tonsillitis, influenza, pneumonia) the bacterial toxins disrupt the chemical processes involved in digestion and thus lower the resistance of the intestinal wall to the bacteria usually habitating the colon; as a result the bacteria penetrate into the small intestine, decomposing the food elements and causing digestive disorders.

Resistance to infections is lowered in premature and feeble infants, in twins and triplets, in babies with congenital heart disease, in neurotic babies and in children suffering from exudative diathesis; these children are easy victims of digestive and nutritional disorders, especially when bottle-fed in the hot months of the year. Hypotrophic and atrophic children are affected with particular severity. Parenteral infections are often observed in child-care institutions where the patients are not given adequate care and strict individual isolation and aseptics are not observed.

Prophylaxis of nutritional and digestive disorders. The most important points in the prevention of these disorders are:

- 1) improving domestic conditions, raising the cultural level of the population, better housing, wide use of fresh air and water, cleanliness of home, clothes, objects used in the care of children, destruction of flies. Practical experience in child-care institutions (crèches, mother and child homes) shows that nutritional morbid-

ity among inmates is low; the same is true of children whose mothers bring them regularly to the district infant health centre;

2) breast-feeding (without additional food) up to six months of age;

3) good milk;

4) proper boiling, cooling and storing of milk (in refrigerators);

5) washing milk bottles thoroughly as soon as the baby has emptied them, and boiling nipples carefully every day;

6) correct management of feeding—no overfeeding, under-feeding or irregular meals;

7) sanitary propaganda among mothers in infant health centres; acquainting mothers with the causes of diarrhea among children;

8) avoiding overheating (stuffy rooms or too warm clothing);

9) giving babies boiled water to drink between meals in the summer;

10) protecting children against infections.

A number of classifications of nutritional disturbances exist, and the following classification is based on them.

CLASSIFICATION OF NUTRITIONAL AND DIGESTIVE DISTURBANCES IN INFANTS AS ADOPTED BY THE 4TH PEDIATRIC CONGRESS OF THE U.S.S.R.

A. Chronic disturbances of nutrition:

1. Hypotrophy

2. Atrophy

B. Acute digestive disorders:

1. Ordinary dyspepsia

2. Toxic dyspepsia

3. Colitis

4. Dysentery

ACUTE DISTURBANCES OF DIGESTION AND NUTRITION

Ordinary dyspepsia usually denominates a generalised disorder manifested by diarrhea, dyspeptic stools, regurgitation or moderate vomiting and extended abdomen owing to the formation of gas. The change in the child's general condition is not too marked, but he becomes a little restless, cries shrilly, his sleep is shorter and lighter and he gains less in weight. Exogenous and endogenous intestinal infections also play a significant part in this, to-

gether with other causes. The acute dyspepsia morbidity rate becomes particularly high in the summer, and mostly among bottle-fed babies. There are several forms of ordinary dyspepsia.

Physiologic dyspepsia. Often the stools of healthy, normally-developing babies is not golden-yellow, but green and more frequent than usual (5-6 times a day) from the very first months of life; this is evidently due to intensive peristalsis of the intestine. The general health and condition of the child are not altered, he is happy, sleeps well, his abdomen is normal, skin pink, turgor satisfactory, weight gain normal.

The cause of this phenomenon is still not quite clear; it probably depends on the digestive peculiarities of such babies, and on the breast milk constituents. No other treatment than observation of the usual rules of hygiene and normal nutrition is needed.

Dyspepsia due to overfeeding. This disorder usually begins with slight restlessness and a more frequent stool. Breast-fed babies can cope with large quantities of food for a long time without any digestive disturbances. By "spitting up" (regurgitating) the baby easily frees himself of superfluous food. In *artificial feeding* toleration of cow's milk is much lower than toleration of breast milk. Notwithstanding this, healthy babies often handle quite large quantities of cow's milk that greatly exceed normal (one litre) requirements, until they finally develop a *digestive disorder*.

It is a well-known fact that many mothers are anxious to fatten their offspring and try to give them as much food as possible. But as we know, too stout children are sick children who contract diseases easily and endure them poorly. Overfed children develop normally until some external factor (overheating, infection) disturbs their equilibrium, evoking nutritional and digestive disorders.

Bottle-fed babies are subject to dyspepsia much oftener than breast-fed babies. Besides quantitative overfeeding, the constituents of the food mixture may also cause dyspepsia: fatty formulas, carbohydrates (chiefly lactose) in large quantities given for prolonged periods, especially in the summer, are poorly tolerated by many babies. Concentrated mixtures should be given in small amounts. In the summer it is better not to prescribe any fatty mixtures at all.

Clinical findings. The child cries shrilly day and night, sleeps poorly, stomach pains make him suddenly cry out loudly. He begins gaining less in weight than formerly. He spits up his milk often, sometimes curdled, sometimes not, at times he has slight

attacks of vomiting, his bowel movements are more frequent, green with small white lumps, looser, and a small amount of mucus appears (dyspeptic stool). The abdomen becomes extended, a large amount of gas is formed owing to the fermentation of the carbohydrates in the intestine under the influence of bacteria. The tongue is coated. Sometimes a slight irregular elevation of temperature is observed.

Prognosis. Favourable for breast-fed infants, while for bottle-fed babies it should be guarded.

Treatment. If the disorder is connected with irregular feeding (at all hours, including the night), or with overfeeding, the gastrointestinal tract is first given a rest for six to twelve hours, depending on the condition of the child; a water diet is prescribed instead of the breast—weak tea without sugar, boiled water or Ringer solution (no more than 300 ml, the rest water alone) in amounts equal to the quantity of breast milk the child receives normally. The mother must express her milk manually. Later the baby is put on a strict nursing regimen (three-and-a-half-hour intervals and a six-hour interval at night) and he is given the breast no sooner than six to twelve hours after the institution of the water diet; the mother should nurse the baby for five minutes at first, and later ten to fifteen minutes not waiting for the stool to improve.

The average duration of the disease is 15 days, in some cases it takes a subacute course and is more protracted. Bottle-fed babies who have been getting large amounts of cow's milk at irregular hours are also put on a six to twelve hour weak tea diet followed by half of the daily milk requirement diluted by half with cereal water and given in six feedings; the liquid deficiency is made up with tea; the dose of milk is increased daily by 100 ml. Besides the usual milk-and-cereal-water mixtures lactic acid mixtures are also prescribed. In severe cases protein milk has a good effect: usually a watery dyspeptic stool turns into a saponaceous fatty stool in the course of several days. Protein milk is given at first in doses of 300-400 ml with 5 per cent sugar in six feedings; the daily dose is increased by 100 ml every day until it attains 200 ml per kg of weight, but is not allowed to exceed 900 ml. A hot-water bag is placed on the baby's stomach. Daily warm baths have a good effect. There is no need to give the child any laxatives. However, some authors recommend castor oil (one half to one teaspoonful depending on age) at the beginning. Astringents (bismuth, tannalbin) are likewise unnecessary as dyspepsia clears up under the influence of diet alone.

Hunger dyspepsia. This is a form of dyspepsia that appears when the mother produces an insufficient amount of milk (hypogalactia). At first the number of bowel movements decreases, the stools become darker (hunger stool); this stage is followed by hunger constipation and the baby has a passage only after an enema. The skin turgor deteriorates, the skin becomes pale. Weight gains decrease, and finally there is no gain at all. The child becomes listless, sleeps a lot, cries after nursing, and soon constipation is replaced by sparse bowel movements, brownish at first, then a green dyspeptic stool with white lumps appears, and the child begins losing weight.

Dyspeptic stools often lead to the conclusion that the child is being overfed instead of underfed. In bottle-fed babies hunger may depend on an excessive dilution of the milk (two-thirds water), an insufficient amount of formula, or a deficiency of certain of the food constituents (carbohydrates, fats). Hungering bottle-fed babies become emaciated sooner than breast-fed babies.

Prognosis is favourable, except for neglected cases of emaciated bottle-fed infants.

Treatment. Treatment consists of prescribing additional food with no preliminary starvation diet. It is often observed that in a matter of only a few days after the baby has been getting one to two additional meals of usual cow's milk formulas, his green stool turns a golden-yellow and he begins gaining weight. For bottle-fed babies the treatment consists of initiating proper dilution of cow's milk corresponding to age requirements (beginning at one month the baby is given C-rice—two-thirds milk and one-third cereal water), a sufficient daily amount of this formula, and cautious addition of carbohydrates and fats. An additional bottle or two of buttermilk with 5 per cent sugar and 3 per cent flour has a good effect; upon improvement of the stool 10 g of 10 per cent sweet cream is added to a 200 ml bottle of buttermilk before sterilisation. Usually the prescription of sufficient amounts of milk lead to good weight gains.

The amount of food is prescribed with consideration for the weight the child should have at his age, not his true weight. One-third to one-fourth less than the necessary amount is given, so that the toleration threshold is not exceeded too suddenly, as that might lead to another severe nutritional disturbance (toxic dyspepsia). In protracted cases of hunger dyspepsia the child's weight does not increase immediately after a normal diet has been initiated (phase of repair). A good weight gain is observed approximately two weeks after the child has been receiving a

fixed quantity of a certain formula; this depends on the restoration of the proper function of the digestive system and of the processes of assimilation.

Dyspepsia in exudative diathesis. This form of dyspepsia is characterised by the appearance of an ordinary dyspeptic stool and regurgitation at the slightest oversight in feeding and caring for the baby; it is accompanied by a sharp fall in weight as the result of a rapid loss of water (hydrolability) and tends to protracted courses. *Treatment* consists of the prescription of 1-2 bottles of protein-rich formulas (buttermilk, protein milk). The amount of fat in the milk formulas given to bottle-fed babies must be decreased, and one to three bottles of buttermilk or protein milk should be prescribed.

Dyspepsia due to intestinal infection. This disease appears when babies are given cow's milk contaminated with virulent strains of *B. paracoli* or *B. proteus vulgaris*, an occurrence not unusual in the summer. The symptoms are the same as for dyspepsia from overfeeding, but its course is much more severe and it often develops into toxic dyspepsia.

Dyspepsia due to parenteral infection. This type of dyspepsia often accompanies generalised infectious diseases in infants (influenza, pneumonia, scarlet fever, measles). Its severity and duration are dependent on the general infection, upon recovery from which the parenteral dyspepsia also clears up. This disorder is more commonly observed in children weakened by preceding gastrointestinal diseases.

Treatment. Treatment of the basic disease and diet as for ordinary dyspepsia.

Toxic dyspepsia is a severe generalised disease characterised by intensive vomiting, loose watery stools, intensive dehydration, weak cardiac activity and acute disturbances of the nervous system manifested by stupor, somnolence, and other generalised brain symptoms. The onset of the disease may be spontaneous and appear at a time when the child seems to be enjoying perfect good health, or it may be gradual, like ordinary dyspepsia. The disease usually occurs in the summer, affecting chiefly bottle-fed infants. Formerly its mortality rate was very high.

Etiology and pathogenesis. The etiology of this disorder has not been studied sufficiently. Among the causes that may lead to it a prominent part is taken by the contamination of the baby's milk and other food, chiefly with the colon bacillus *Escherichia coli* (P. Medovikov, G. Speransky, I. Tzimblor). It is, moreover, not improbable that the microbes of the child's intestinal tract may become activated (*proteus*, *enterococcus*, *B. perfringens*) under the

influence of altered environmental conditions (overheating, chronic nutritional disturbances, previous illnesses); these hitherto harmless microbes become pathogens, evoking toxic dyspepsia.

The mechanism of infection in toxic dyspepsia still awaits its final clarification.

Disturbances in surrounding environment are sources of excessive irritation in young children. Intoxication caused by the colon bacillus or the products of the incomplete disintegration of food are sources of overexcitation of the child's central nervous system; the lability and reactivity of the latter decrease, the functions of all the organs and systems become impaired.

As a result of the changes in the reactivity of the central nervous system and of a resulting dysfunction of the liver the child's metabolism becomes disturbed, vitamin deficiencies (hypovitaminosis) develop, resistance (immunity) is lowered, and all this leads to the development of a severe form of dyspepsia—toxic dyspepsia. Therefore, the clinical findings in toxic dyspepsia show a prevalence of generalised manifestations of a severe toxicosis connected with primary lesions in the central nervous system.

Clinical findings. The temperature is often elevated from the very onset of the disease (up to 39°). At the beginning frequent projectile vomiting of large portions of food is observed; subsequently the vomitus becomes sparser, containing admixtures of bile, in the most severe cases it has the appearance of coffee dregs speckled with blood. The stool is frequent (10-20 times a day) and watery; it contains particles of greenish feces and small amounts of mucus. The child's lips are dry, his tongue white-coated, the mucous membranes dry, in some cases thrush or ulcers are observed in the mouth. The abdomen becomes distended owing to excessive gas-formation, sometimes increased peristalsis is noted. The fontanel may be depressed owing to excessive dehydration and exsiccation. The deep-sunk, half-closed eyes of the child fix their suffering gaze on one point. The toxic spasm of the vasomotors causes the skin to become pale-grey. The turgor of the skin decreases greatly: pinched into folds it smoothens out very slowly. Muscular tone at first decreases, while later on hypertonia may develop. A characteristic position of the upper extremities is often observed: bent-in elbows and fingers, the so-called "fencer's hand". Sometimes automatic motions of the arms are noted ("music conductor's hands"), indicating irritation of the cortical and subcortical layers of the brain; the extremities are cold and cyanotic. Sclerema sometimes appears on the legs shortly before death.

A sharp change is noted in the general condition: somnolence, apathetic expression, stupor, sudden outcries, mask-like face, sometimes convulsions ("dyspeptic coma"). The heart sounds become dull, the first sound sometimes disappearing. The pulse is

irregular, small, and at times the discrepancy between the pulse and cardiac activity is noticeable: the pulse is still normal, while the heart sounds are already almost obliterated. This probably depends on extension of the lungs. Breathing becomes irregular, (the Cheyne-Stokes type of respiration), sometimes deep accelerated respirations are noted, up to 50-60 per minute (stertorous or "panting" respiration). This type of breathing may be explained by the acute intoxication of the organism and excitation of the respiratory centre. Postmortem examinations usually show no inflammatory seat in the lungs that might have been the cause of this panting breath. The rapid loss of fluids and emaciation, forceful vomiting and diarrhea evoke a sharp fall in weight (100-200 g and more a day), and folds appear in the skin over the thighs. The blood thickens with dehydration and this leads to elevations of hemoglobin content and erythrocyte count (hyperglobulinemia), and to neutrophilic leukocytosis (up to 15,000-20,000 neutrophils). Exsiccation leads to a sharp decrease in the frequency of urination; the total amount of urine is greatly reduced, in some cases the child scarcely urinates at all, as the entire fluid is depleted with the vomitus and through the bowels, and also through the skin and lungs. In these cases mothers often ask the doctor to "drain the child's bladder", as he has not passed water for a whole day; however, the bladder turns out to be empty. Frequently urinalysis shows albumin, hyaline and granular casts, single erythrocytes, and up to 15-30 leukocytes per field of vision (pyuria). Often sugar also appears in the urine.

Duration. The disease lasts from two to four weeks. Protracted cases are also encountered in connection with various complications, lasting one and a half to two months. The disease may be mild or very severe. In typical cases diagnosis is not difficult. *Complications* affecting the ears are often observed: acute inflammation of the middle ear, insidious mastoiditis (inflammation of the mastoid antrum). The ears of toxic dyspepsia patients should be examined frequently. Early diagnosis of mastoiditis and proper treatment save the child's life. Head-rolling and uncontrollable vomiting accompanied by slight diarrhea, slight rigidity of the neck, toxicosis, fever, leukocytosis, as well as X-ray examinations of the mastoid process and antral puncture are factors to be considered in determining the diagnosis. Complications aggravate the course of the disease. Inflammation of the middle ear is observed quite frequently in infancy. According to M. Skvortsov, post-mortem examinations show otitis in about 80 per cent of lethal cases in infancy. Pneumonia is often also observed, as are thrush,

ulcers in the mouth, and keratitis due to the dehydration of the cornea. In severe cases, where the patients are emaciated and not properly cared for, *pyodermatitis* appears (see *Skin Diseases*). Another complication may be a severe attack of pyelitis with a large amount of pus in the urine; this may also be discovered during autopsy; often a more or less pronounced nephritis is also present.

Prognosis is grave. The earlier the disease is recognised and treatment instituted, the sooner the child is hospitalised—the better the prognosis.

Prophylaxis consists of nursing babies on the breast, starting cereals no earlier than at six months, supplying children with good cow's milk, and conducting sanitary propaganda among the population to acquaint mothers with the causes of this severe illness. Among breast-fed infants this disease is observed less frequently than among bottle-fed babies. However, breast-fed babies can also be affected by it when rules of hygiene are grossly violated, and when cereals are given in addition to the breast in the very first weeks after birth. The principal factors in the prevention of toxic dyspepsia are breast-feeding with no additional solids before six months, good care, cleanliness, fresh air and precautions against overheating. Emaciated (hypotrophic) children and children reconalescing from various other illnesses are most easily affected.

Treatment. A 12 to 24 hour water diet is prescribed from the very beginning (or weak unsweetened tea), or Ringer solution (no more than 200-300 ml, the rest water or tea) in quantities corresponding to the child's age (as much as the child had been getting when healthy). The fluid is given cold, 2-3 teaspoonfuls every ten minutes, or by drops with a medicine dropper. After 24 hours 50 ml of expressed breast milk is prescribed for a day, in ten doses of 5 ml each, also cold; the amount of milk is increased daily by 50 ml until it reaches three-fourths of the normally required amount; water is given simultaneously, to make up the full amount of fluid required at the given age (for instance, 700 ml of milk and 200 ml of water for a three-month baby).

This quantity is given for a week. Greater amounts of milk might aggravate the disease. Sometimes the infant does not take undiluted breast milk readily; in these cases it is recommended to add several tablespoonfuls of milk to a glass of water, and the child will take it more readily.

Some authors recommend skimmed breast milk. The milk is put on ice or in cold water for four hours, and then the cream

is removed; if the mother has no milk of her own, she gets it at the milk banks of the infant health centres, in maternity hospitals, or from any nursing mother (this milk should be boiled). If there is no way of getting breast milk the baby is given separated cow's milk, protein milk, or buttermilk diluted by half with boiled water. Protein milk and buttermilk are given according to the same method as breast milk, but these mixtures are not always tolerated by infants, and they frequently evoke vomiting. If a breast-fed infant does not vomit for five days he may be nursed at the breast for five minutes, once a day, during the third or fourth feeding. If vomiting recurs a water diet is again prescribed for 12 hours, and then frequent small doses of milk are given, less guardedly than before (adding about 100 ml daily).

At the beginning of the disease gastric lavage may be beneficial. Laxatives should not be administered, as they only increase vomiting and diarrhea. A low pulse calls for caffeine, 1 per cent solution, one teaspoonful three times a day, or subcutaneously in a 10 per cent solution (Sol. Caffeini natrio-benzoici 10 per cent, Sterilis), three times a day in 0.25 ml doses.

Hot-water bottles are put to the feet. Warm baths (36-37°C) are also beneficial, for five minutes daily. Petroleum jelly (vaseline) should be applied to the lips. The eyes are washed with boiled water, and drops of sterilised mineral or fish-liver oil are instilled to prevent the cornea from drying. Utmost cleanness of the skin, particularly in the perineal area, is very important. Fatty substances are used for softening it, and boiled vegetable or mineral oil is applied to the diaper area after it has been washed. The application of fats and oils should be performed very gently in order to avoid abrasion of the skin. The child is placed on a special rubber bed-ring to prevent the formation of bedsores, and he should be turned from side to side. The sacral region is sponged with 20° alcohol. A diaper should be tied around the neck, to receive the vomitus, as it is easily removed. The room must be aired as often as possible, and overheating avoided by all means. In cases of severe vomiting and great weight losses luminal has a good sedative effect, and also subcutaneous infusions of Ringer solution (100-200 ml daily into the subcutaneous tissues of the abdomen; contraindicated in sclerema).

The possibility of intestinal infection is not excluded in toxic dyspepsia. Therefore, antibiotics are used in its treatment (as for dysentery), such as synthomycin and levomycetin (chloramphenicol); besides, it is often difficult to be certain that the infant has

not got dysentery accompanied by toxic manifestations very similar to those of toxic dyspepsia.

In toxic dysentery emaciated children are usually put on a hunger diet for six hours, sometimes for longer, but for no more than 12 hours. In severe protracted cases of toxic dyspepsia a good effect is sometimes obtained by blood transfusion (5-10 ml per kg of weight). Two or three transfusions are given over five-day intervals. Mastoiditis patients are given ordinary doses of sulfazol for ten days, and simultaneous penicillin injections (see *Sepsis of the Newborn*), and blood transfusions. If this treatment is unsuccessful antrotomy is performed. Other complications are treated symptomatically (see sections on pneumonia, stomatitis, pyoderma).

Summer diarrhea. The following diseases are known under the name of summer diarrhea: ordinary dyspepsia, toxic dyspepsia, dysentery. These types of diarrhea are particularly frequent in the summer, and the hotter the season, the higher the diarrhea rate.

However, these diseases are likewise encountered in the winter, but much less frequently.

Prophylaxis of summer diarrhea consists of the following: breast-feeding (without additional solids) up to six months, if possible; the baby should not be weaned from the breast in the summer; transfer to cow's milk must be very guarded and gradual. Children should be supplied with good cow's milk. Every measure must be taken against overheating (if possible, the baby should be kept in the open air from morning to evening; windows should be open day and night; light clothing, air baths several times a day, the child's body sponged with tepid water, boiled water offered between meals—a glass a day). Flies should be destroyed (window nets, fly-paper, clean house and yard). Clean clothing, clean objects used in the care of the child, his body kept clean (daily baths and rub-downs). As many children as possible should be enrolled in crèches; special summer crèches should be opened. The staff of the infant health centres should keep a special register for children most easily afflicted by summer diarrhea, and these children should be given priority in receiving full value milk and nutritive mixtures (bottle-fed, emaciated, atrophic, or premature infants, babies recuperating from severe illnesses and nutritional disturbances, or just dismissed from hospitals). Wide-spread sanitary propaganda in the infant health centres and crèches (lectures for mothers to acquaint them with the causes and prevention of summer diarrhea); wide propagation of printed leaflets and booklets on summer diarrhea, lectures over the radio.

For *treatment* see corresponding chapters.

CHRONIC DISTURBANCES OF NUTRITION

Hypotrophy. Hypotrophy is a generalised chronic nutritional disturbance accompanied by slow weight gain or arrested weight, or even losses due to various causes evoked by lesions of the mesenchyme.

The *causes* of hypotrophy are: more or less prolonged starvation owing to insufficient production of milk by the mother, or to excessive dilution of cow's milk; monotonous diet of cow's milk exclusively, leading in certain cases to the nonassimilation of milk and the appearance of a saponaceous fatty stool; exclusive carbohydrate diet (grain flour, cereals, bread) that leads to brownish watery stools and muscular hypertonia; severe, acute, subacute and chronic infections (pneumonia, pyelitis, tuberculosis, syphilis); severe acute digestive disorders (during toxic dyspepsia, dysentery, etc.); protracted parenteral digestive disturbances; bad care, stuffy rooms, antisanitary conditions; a disturbed balance of proteins, carbohydrates and fats; vitamin deficiencies, often rickets. The disease is most frequently observed in the first year of life.

Clinical findings. Hypotrophy is manifested first of all by the baby's normally pink skin becoming pale, and its turgor deteriorating. Subsequently, as the disorder aggravates, folds appearing in the skin over the thighs indicate the diminishing subdermal layer of fat. Muscular tone first decreases, and later muscular hypertonia appears. The weight gain curve becomes flatter, but for several days it still goes upward, then it goes down and stabilises; thus, the weight curve of hypotrophic children is irregular and wavy. Later on the child's resistance deteriorates: hypotrophic children are easier subjects for nutritional and digestive disorders, influenza (grippe), pneumonia, and pyoderma.

The stool often remains normal, at times it becomes more frequent, rarer—less frequent. When cow's milk is not assimilated properly the stools retain their form, but become of a whitish soapy-fat consistency; an exclusive carbohydrate diet causes the passage of frequent brown stools. Digestive disorders are observed frequently in hypotrophic, and particularly atrophic children; their course is severe, tending to protracted periods. In the absence of proper treatment protracted digestive disorder subsequently develops into a grave form—atrophy. In acute and chronic infections the diagnosis may be determined easily by the symptoms of the acute or chronic infection, and thus the cause of the hypotrophy established and proper treatment instituted; urinalysis should

never be omitted, as a latent pyelitis is often the cause of hypotrophy.

Hypotrophy may be of several degrees of severity. Three degrees are known.

Hypotrophy of the 1st degree. Weight deficiency is 10-20 per cent of the child's normal weight. The subdermal fat layer decreases over the abdomen alone. Tissue turgor is only slightly decreased.

Hypotrophy of the 2nd degree. Weight deficiencies exceed 20-40 per cent of the child's normal weight. The subdermal layer of fat decreases more significantly, not only over the abdomen, but also over the rest of the body and the extremities. However, this is not very noticeable on the face.

Hypotrophy of the 3rd degree (atrophy). This is the severest form of a chronic nutritional disorder, combined with extreme emaciation. The child's skin becomes earthy-grey. No subdermal fat remains at all, the so-called fat lump (Bichat) in the cheeks disappears, and the face becomes old and wizened. Next numerous folds appear in the skin over the thighs. Skin turgor and muscular tone decrease, the abdomen is often depressed, the temperature falls sharply (sometimes below 36°), the pulse slows down, respiration becomes irregular and slower. Resistance to infections falls sharply. These patients are often afflicted by pyoderma and pneumonia, frequently at normal temperature levels. The feces sometimes remain normal, in some cases a saponaceous fatty stool appears, infrequently vomiting is observed. The duration of the disease is extremely protracted.

The assimilation of food decreases sharply; any, even moderate, increase in its volume leads to a loss in weight and aggravation of the general condition (paradoxical reaction), while, on the other hand, small amounts of food also result in loss of weight. Thus, the amount of food that can be tolerated by the patient has to be established for each individual case.

Prognosis depends on the basic cause of the disease. The earlier treatment is initiated, the more favourable is prognosis.

Prophylaxis of hypotrophy and of its severe form, *atrophy*, consists of proper management of feeding, timely addition of expressed breast milk or formula if the mother's supply fails.

The child must be given all the necessary vitamins (fish-liver oil, fruit juices).

A proper sanitary and hygienic regimen must be established for the prevention of diseases (clean premises, taking the baby outdoors). Contact with sick children should be avoided.

All hypotrophic children should be specially registered at the infant health centre or polyclinic in order to provide timely medical aid and prevent the development of atrophy.

Treatment consists of eliminating the causes of hypotrophy. If the child has been hungering, he should be given additional food—ordinary formulas or buttermilk with 5 per cent sugar and 3 per cent flour, kefir with 5 per cent sugar and 5 per cent cream, lactic acid formulas and acidophilus milk. It has been found beneficial to add one-half to one teaspoonful of plasmon to the mixtures (before boiling them). If the child is fed exclusively on milk and does not assimilate it, skim milk mixtures with 5 per cent sugar and 3 per cent flour and vitamins (fruit juice) are prescribed. Upon the disappearance of the saponaceous fatty stools the child is gradually transferred to mixtures of whole milk with flour. If the baby has been getting only specially prepared children's flour, bread and cereals the usual milk formulas with cereal water are prescribed, also 1-2 bottles of buttermilk with 5 per cent sugar and kefir with 5 per cent sugar and 5 per cent cream. In severe cases it is necessary to replace at least half of the food by breast milk.

With these mixtures the children usually commence gaining weight. Sometimes the weight remains stable for ten to twenty days (phase of repair), and only after this stage does the child begin to take on weight on the same amount of formula. Besides this, intramuscular injections of maternal blood or serum are given for ten days, 10 ml daily; a satisfactory weight gain is often marked. A good effect is also obtained by the injection of citrated blood into the antecubital vein or one of the veins of the scalp after determination of the child's blood group (10 ml of citrated blood per kg of weight). Campolon injections (10-15 in all) are also effective, in daily doses of 1 ml into the thigh muscles. The amount of food given to hypotrophic children should be increased gradually to 130-150 calories per kg of weight.

It is also important to keep the child's skin clean, give him warm baths every day, keep him in the fresh air as much as possible, and give him vitamins (fruit juices and fish-liver oil).

In some cases gastric juice (one-fourth to one-half teaspoonful twice a day) is given before meals, or a 1 per cent solution of diluted hydrochloric acid (*Ac. muriatici diluti*) with pepsin (one teaspoonful three times a day) and after meals pancreatin (0.1-0.15 g twice a day). Massage, physical exercises and fresh air are general strengthening and health-promoting factors.

PYLORIC STENOSIS

Pyloric stenosis or *pylorostenosis* is a congenital thickening of the pyloric musculature owing to defective development in fetal life. Besides this thickening, the pylorus is constricted. The disease is accompanied by vomiting, constipation and a more or less marked emaciation. It is most often manifested in the third week of life, beginning with projectile vomiting to which constipation is added. In the first few days of life there is no vomiting, owing to the small intake of milk and the strong muscles of the stomach; however, as the amount of milk intake increases and the stomach musculature weakens with the extension of this organ, the food can no longer pass through the hypertrophied constricted lumen of the pylorus, and this results in projectile vomiting. Weight begins falling, and often such a child weighs less at 5-6 weeks than he did at birth. The flesh wastens, the forehead wrinkles and the child acquires a characteristic pale and sullen aspect. The *skin* becomes pale, numerous folds appear over the thighs and hips. In most cases *gastric waves* are evident in the *substernal area* in the shape of an hourglass. In some cases the eyes are deeply sunk, stupor and somnolence are observed (*pyloric coma*). Vomiting becomes progressively more forceful, the mass of vomitus greatly exceeding the amount of milk taken in. The disease is more common in boy infants than in girls, and is less frequently observed than pylorospasm. Undiagnosed and untreated cases have rapid, fatal terminations. Many authors look upon pylorospasm and pyloric stenosis as one and the same disease of various degrees of severity, and consider that pylorospasm occurs much more frequently than pylorostenosis.

Differential diagnosis between pylorospasm as a purely functional disorder devoid of organic defects in the pylorus, and pyloric stenosis is established by the presence of the following symptoms:

Pylorospasm

- a) Neuropathic mothers in almost all cases.
- b) Vomiting from the moment of birth.
- c) Frequent vomiting.
- d) Frequency of vomiting extremely variable on different days.
- e) Lesser volume of vomitus mass.
- f) Amount of milk in vomitus less than milk intake.

Pyloric Stenosis

- a) Mothers rarely neuropathic.
- b) Vomiting commonly begins two weeks after birth.
- c) Less frequent vomiting.
- d) Frequency of vomiting more uniform.
- e) Projectile vomiting in large amounts.
- f) Amount of milk in vomitus more than milk intake.

- g) Constipation, but the child has bowel movements at times.
- h) Frequency of urination decreased (approximately 10 times a day).
- i) Skin not very pale.
- j) Gastric waves rarely observed.
- k) Child restless, cries loudly.
- l) No weight gain, or moderate loss in weight.
- g) Severe constipation in almost all cases.
- h) Frequency of urination sharply decreased (approximately 6 times a day).
- i) Skin very pale, striated and in folds, wrinkles on forehead.
- j) Gastric waves observed very frequently, sometimes in the shape of an hourglass.
- k) Child quieter.
- l) Sharp loss in weight.

X-ray findings. Barium added to the infant's food (expressed breast milk) allows the demonstration of prolonged retention of food in the stomach (over 4-6 hours) in cases of pyloric stenosis, while in pylorospasm the stomach is empty of food after three to four hours.

Prognosis is grave. Early diagnosis and surgical repair save the infant's life, giving complete relief.

Treatment. Frequent (every two hours) or rare (five times a day) feedings are recommended; the food is either warm or cold breast milk deprived of fat, concentrated formulas (breast or cow's milk with 17 per cent sugar added), buttermilk with 5 per cent sugar and 3 per cent flour, nutritive enemas consisting of breast milk and 17 per cent sugar, cereal cooked on breast or cow's milk (one teaspoonful before usual feedings). However, all these methods of feeding were not found to be successful in pyloric stenosis. The administration of solutions of atropine (1:1,000), one to five drops three times a day, sometimes even until the pupils dilate, also gave no beneficial results.

The effective method of treatment is pylorotomy (longitudinal splitting of the muscular layer of the pylorus, leaving the mucous layer intact). Prior to surgery the infant is given no milk for three hours, an hour before the operation gastric lavage is performed, in severe cases 100 ml of Ringer solution with an equal quantity of a 5 per cent solution of glucose is injected subcutaneously into the thigh; if a small soft pulse is observed a subcutaneous injection of caffeine or camphor is indicated. Blood transfusions are recommended pre- and postoperatively, particularly if the child is markedly debilitated.

After the operation the child is placed in his crib, supported on both sides by sheet rolls; hot-water bags are placed at the feet, while a towel is passed over the chest and secured to the edges of the crib. Six hours after surgery feeding is recommenced: 30 ml of breast milk ten times in 24 hours, every two hours, adding

100 ml daily. Three days after surgery the child is given the breast (the mother bending over him) once a day for five minutes. Further the amount of milk is increased daily, and the infant is gradually transferred to breast-feeding. The child is weighed only on the seventh day. The result of the operation depends to a large extent on good nursing. In some cases vomiting and constipation disappear directly after surgery, and the baby's weight gain may be 200 to 300 g in the very first postoperative week. In a minority of cases a gradual disappearance of vomiting is observed.

As a rule operated infants improve. Subsequent medical examinations of such stenotic patients have shown that the symptoms of the disease disappeared and that there were no recurrences.

CONSTIPATION

Children are often constipated in early infancy. This constipation may be due to a number of causes: 1) insufficient supply of mother's milk, highly diluted cow's milk or an insufficient daily quantity of formula (hunger constipation); 2) excessively fatty breast milk or a too large amount of fatty formulas or whole milk; as a result of the high protein content in cow's milk; 3) atonia of the intestine and the intestinal wall, as in rickets and other diseases (atonic constipation); 4) anal fissures (owing to pains on attempted evacuation); 5) congenital constriction of the pylorus or of the small intestine; 6) congenital dilation of the duodenum or small intestine; 7) adhesive tuberculous peritonitis; 8) acquired intestinal constriction (the small intestine or the rectum); 9) strangulated hernia.

Prognosis depends on the causes of constipation.

Treatment. 1. Additional food, increasing daily volume of food and carbohydrate formulas: buttermilk with 5 per cent sugar and 3 per cent flour added, one-day kefir with sugar, large quantities of fruit juices (50 ml a day). 2. If the breast milk is too fat the mother's diet should be modified by large quantities of vegetables and fruits, while the infant is given a solution of lactose (one teaspoonful to a cup of water) 5-6 times a day, or fruit juices. 3. Spastic constipation is dealt with by general strengthening measures, warm baths with pine extract, large quantities of fruit juices. Rectal suppositories with belladonna are used. Some authors recommend the application of diathermy to the abdomen (not exceeding 0.5 mA, three to five minutes every day). 4. Atonic constipation is successfully treated by general strengthening measures, such as saline baths, massage of the abdomen, physical

exercises for strengthening the abdominal muscles, one-day kefir with cream, fruit juices and fish-liver oil. 5. Fissures of the anus are treated with a 5 per cent solution of silver nitrate and a vegetable diet with a high fat content (1-3 teaspoonfuls per day) to soften the feces, as hard stools inflict pain upon passage and are a cause of constipation. 6. In congenital or acquired intestinal constriction surgery is indicated. 7. Tuberculous peritonitis is dealt with in the corresponding chapter. As a measure of temporary relief in constipation glycerin suppositories are inserted at night, and enemas are given with glycerin (one teaspoonful to a glass of water), or pure vegetable oil (2-3 tablespoonfuls) followed by a purging enema. However, enemas should be given cautiously, as the intestines must be trained to act independently.

One teaspoonful of purified mineral oil is given three times a day orally for one to three weeks for chronic constipation.

PATHOLOGIC CONDITIONS DUE TO VITAMIN DEFICIENCIES

For its proper growth and development the body requires not only proteins, fats, carbohydrates, water and minerals; the normal course of physiological processes also calls for special additional substances supplied with food in minute amounts. These substances are vitamins.

The absence of vitamins in food, or their failure to be assimilated in the intestinal tract and thus to be delivered to the tissues of the body lead to the development of vitamin deficiencies, both generalised (growth disturbances, general flaccidity, loss of appetite, irritability, sleeplessness), and peculiar to each vitamin. *Hypovitaminosis* is observed much more frequently; this is a condition due to a deficiency of vitamins in the tissues.

It is commonly considered that the principal cause of hypovitaminosis in man is nutritional failure due to an insufficient intake of vitamins with food, and at the same time a deficiency of other dietary ingredients (proteins, fats). Hypovitaminosis is sometimes observed in periods of intensive growth in children, and in women during pregnancy and lactation, when the usual supply of vitamins is insufficient, and a greater amount is required.

Various diseases may likewise lead to hypovitaminosis. Thus, tuberculosis, rheumatism, pneumonia, dysentery and other illnesses are accompanied by a marked vitamin deficiency; many diseases call for the introduction of additional quantities of vitamins into the body. In gastrointestinal diseases, when the vita-

mins contained in the food are not assimilated, it is often necessary to introduce vitamins parenterally (oral administration is useless); they are given in subcutaneous, muscular or intravenous injections. Often the organs are deficient in a whole group of vitamins, not in one vitamin alone. Such a condition is called *hypovitaminosis*. In some cases hypovitaminosis in children is not markedly manifested, and this condition is defined as a "smudged" form of vitamin deficiency.

Avitaminosis and hypovitaminosis do not become manifest immediately after the cause of the avitaminosis or hypovitaminosis has begun acting, but only after some time has elapsed. The reason of this is that the store of vitamins in the organs and tissues (chiefly in the liver) does not become depleted immediately, and these accumulations maintain a definite vitamin level in the tissues and the blood for a more or less protracted period.

PATHOLOGIC CONDITIONS DUE TO VITAMIN A DEFICIENCY

Vitamin A plays an extremely important part in the vital activity of the child's body. It is a factor promoting resistance against infections, protecting the epithelium against degeneration and making it work properly. Vitamin A raises the antitoxin level in the blood and is therefore given in all infectious diseases.

Keratomalacia is a vivid illustration of the harmful effect of the absence of vitamin A on the epithelium of the cornea.

The symptoms of vitamin A deficiency are: night blindness (*nyctalopia*) when vision in the dark is greatly reduced owing to a deficiency of vitamin A in the retinal purple of the protein compound; xerosis of the conjunctival epithelium and the epithelium of the eyelids; dryness of the eye, partly owing to changes in the epithelium, partly to decreased secretion of tears; xerophthalmia (Fig. 20) due to cornification of the epithelium; subsequent purulent softening of the cornea (*keratomalacia*) and loss of the eye; decreased ability to discern blue and yellow in the daytime; loss of olfactory sense due to lesion of the olfactory region; the development of a fetid discharge from the nose—*ozena* or *orhinitis sicca*; the appearance of a ridge



Fig. 20. Hypovitaminosis
A. Xerophthalmia (leukoma on an eye)

on the line between the skin and mucous membrane of the eye, nose and mouth; cracks in the corners of the mouth; the development of persistent aphthous or ulcerative stomatitis, and also very stable thrush covering the entire buccal mucosa, gums, tongue and throat; respiratory symptoms are hoarseness due to changes (metaplasia) in the epithelial lining of the larynx, persistent bronchitis, protracted pneumonia; in the digestive sphere there is an irregular growth of the teeth with excessive enamel and cementum formation, lowered gastric juice acidity attaining in some case complete achylia; persistent diarrhea, loss of appetite; in the urogenital sphere abundant desquamation of the epithelium of the bladder, urethra and vagina; formation of calculi in the bladder and kidneys; nonspecific (nongonorrheal) urethritis; failure of the function of the adrenals accompanied by a fall in blood pressure; the skin is dry, pale, scaly and pigmented owing to lesions of the epithelium and lowered activity of the sebaceous and sweat glands; the skin of the palms and soles is inflamed and red, long ridges appear on the nails, they become brittle and often crumble; paronychia and panaritium appear easily; in rare cases partial necrosis of the skin occurs on the soles, the hair loses its lustre, becomes dry and greys early; besides this, there is a marked emaciation and loss of flesh; the skin hangs off the thighs in folds.

Naturally, in every individual case there exist only a part of the above symptoms. In hypovitaminosis the symptoms are less pronounced. When the child begins taking milk, fresh butter and fish-liver oil regularly all the manifestations gradually disappear. Only the most severe cases are mortal. To avoid eye lesions fish-liver oil is instilled into the eyes of all seriously ill patients, as well as of patients with affected cornea. Timely administration of fish-liver oil or fresh butter often saves the child's vision. As a preventive measure children should always have milk, butter and fish-liver oil (one teaspoonful or one tablespoonful twice a day, depending on age), as well as carotene and fresh vegetables. Infants must not be kept for too long a period on buttermilk and protein milk, as the vitamin A and fat content of these substances are low. It must also be remembered that there is less vitamin A in drawn butter than in fresh, and none in pork lard. In breast-feeding and mixed feeding the mothers should take vitamin A in their food twice a day in 10 to 15-drop doses for 2-3 weeks.

PATHOLOGIC CONDITIONS DUE TO VITAMIN D DEFICIENCIES

In the child's body vitamin D is formed from provitamin D. This transformation takes place in the skin when it is irradiated by sunlight or ultraviolet rays.

A healthy child gets the necessary amount of vitamin D chiefly by synthesising it in his own body, and to a much lesser extent from his food. Vitamin D is contained in milk, in fish liver and oil, in caviar (fish roe) and in egg yolk.

A deficient formation of vitamin D in the child's body or its low assimilation, particularly when its content in the food is not high, leads to rickets.

Rickets

Rickets is a chronic generalised disease characterised by altered mineral metabolism and typical deformations of the bones that is most commonly observed from one to 18-24 months of age. It is accompanied by varying degrees of anemia, relaxation of ligaments and poor development and tone of muscles, enlargement of liver and spleen, neuropathies, and also tendencies to slowly developing protracted disorders of the respiratory tract and prolonged diarrhea followed by constipation.

It is mostly children living under bad conditions and raised on cow's milk who are affected by rickets.

Etiology. Vitamin D deficiency is the primary principle in the etiology of rickets. There are a number of factors that facilitate the development of vitamin D deficiency in the tissues of the child's body, and the disruption of normal metabolic processes.

Living conditions: rickets mostly affects children who live in damp, dark premises where there is not enough fresh air nor sunshine; rickets also affect children who live in good houses when their rooms are stuffy and badly ventilated, and they are not out in the fresh air enough in the autumn and winter; *nutrition:* breast-fed babies are less apt to have rickets than babies raised on cow's milk; the latter often have rickets, as boiling milk destroys its *vitamins* and other ingredients necessary for the proper development of the child; *seasonal conditions:* rickets is much more frequent in the winter when children do not get enough fresh air and there are not enough ultraviolet rays in sunlight; the beginning of rickets is rarely observed in the summer months; *climate:* rickets is most widespread in countries with temperate climates;

it is less frequently encountered in southern countries; at higher altitudes rickets is also less likely to occur owing to the intensity of ultraviolet radiation in the mountains; premature infants, twins and triplets are more apt to get rickets; *acute, chronic diseases, grave acute infections*, as well as severe nutritional disorders often aggravate rickets.

Pathologic anatomy and pathogenesis of rickets. The nature of the development of rickets is the lowered ability of the cartilaginous cells to deposit calcium (leading to brittleness of the bones), and also excessive formation of osteoid tissue in which there is almost no deposition of calcium; this tissue forms rachitic beads and bossing (knobs on the parietal and frontal bones of the skull).

Metabolic disturbance in rickets is manifested by a sharp fall in the blood phosphorus level (from the normal 5 mg per cent to 3.1 mg per cent), while the calcium content in the blood may be normal or only slightly lowered (6.8 instead of 10 mg per cent). Thus the calcium deficiency in the bones is not the result of an insufficient supply, but is due to a sharp failure of the cells to deposit calcium in the bones, and to an alteration in the calcium and phosphorus metabolism and ratio. Rachitic bones are significantly poorer in calcium and phosphorus than normal bones. The osteoid tissue either does not become calcified at all, or does only to a very small extent. Upon recuperation an intensive deposition of calcium occurs in the newly formed excessive osteoid tissue.

Clinical findings. The earliest symptom of rickets is a nervous disorder displayed by fretfulness, shrill crying and poor sleep. The mother reports that baby resents being "held by the sides"—that is, the bones have become painful to the touch. At about three months after birth the child's head begins perspiring (wet pillow) and bald spots appear on the back of the head as a result of rubbing against the pillow. Upon palpation of the skull the occipital bone is found to be softened to a certain extent as a result of the depletion of calcium (craniotabes). It must be remembered that premature infants may have a so-called "soft skull" at birth, but that this is no symptom of rickets.

Softening of the occiput may continue for several months in the absence of proper treatment. Besides the softening of the occiput, knobs appear on the frontal and parietal bones (square head or saddle-shaped cranium) as the result of an excessive formation of osteoid tissue. Deficient ossification results in the large fontanel growing bigger after six months instead of becoming smaller, and not closing until as late as two years. Rickets is often attended by a slight edematous swelling of the head that gradually disappears upon recovery. The earliest symptom observed in the thorax is the appearance of costochondral prominences (*rachitic rosary* or beads) on the ribs; these beads may attain the dimen-

sions of a hazel-nut and be present for a long time. The softening of the bones and weakening of the musculature lead to lateral depression of the chest and a prominent sternum—*pigeon (or chicken) breast* deformity. Protrusion of the abdomen deflects the sides of the thorax upward leading to flaring of the chest.

The baby's weak joints and muscles cannot hold up his weight when he begins sitting and standing up, and therefore a rounded rachitic hump (kyphosis) may easily develop in the lumbar or thoracic part of the spine (Fig. 21); this is usually the case when rachitic children begin sitting up too early. A baby that is habitu-



Fig. 21. Spinal curvature in rickets

ally carried on one arm in a sitting posture at a too early age easily develops scoliosis (lateral curvature of the spine). However, the rounded rachitic kyphosis should be distinguished from the angular tuberculous curvature; when a rachitic child is placed on its stomach and lifted by his legs rachitic curvatures straighten out, while tuberculous kyphosis does not. Rachitic deformities of the pelvis are observed chiefly in cases when the baby has been allowed to sit up too early (flat pelvis, increased transverse and decreased longitudinal dimensions). In subsequent life this deformity in female babies may play an important part in the mechanism of child-birth. Alterations in the extremities: widening of the epiphyses of the ulnar and

radial bones in the wrist (rachitic bracelet); owing to the thickening of the shafts of the digits the fingers take on a "string of pearls" appearance. In cases of lowered muscular tone the child's softened long bones bend under the weight of his body, and he becomes bow-legged. Moreover, bow-legs also occur because the ends of the soft bones are pulled inward by the stronger flexor muscles; knock-knees are observed less frequently owing to the weakness of the ligaments in the knee-joints and the natural slight outward arching of the lower limbs. The thigh-bones may also become bent. Green-stick fractures and fractures of the long bones are not uncommon among rachitic children; dentition is retarded, and the

order of eruption is not as in healthy babies. The relaxation of the ligaments may be of such a degree that the joints become loose, leading to deformities. The tone of the striated musculature is significantly lowered (hypotonia); a simultaneous weakening of the smooth muscles evokes extension of the intestines. The weakened muscular wall of the abdomen cannot sustain the pressure of the gas-inflated intestines, and the result is a large protruding abdomen ("frog belly").

Static functions are very retarded in rachitic children. In some cases the child only starts walking at two years. As rachitic children are more or less anemic they are usually pale (see chapter *Diseases of the Blood*). In rickets the liver and spleen are often moderately enlarged, their edges remaining soft. Digestive disorders accompanying rickets are of a dyspeptic nature and are very persistent, being replaced at times by prolonged constipation. Immunity is decreased in rachitic children, so they easily contract influenza, measles, whooping cough, illnesses with severe courses and frequent complications (pneumonia). Whooping cough and influenza are often very protracted.

In severe cases of rickets the general state of the child is seriously impaired.

Rickets usually continues for many months (particularly if untreated) and often leaves sequences for life in the form of curvatures of the spine, extremities or chest.

Classification of rickets (as accepted by the 6th National Congress of Soviet Pediatricians in 1947). At present the forms of rickets are distinguished by three basic factors: 1) stage; 2) course; and 3) severity of the disease.

The following is the currently accepted classification of rickets.

Stage of Disease	Course	Severity
Initial	Acute	1st degree, mild
Greatest intensity	Subacute	2nd degree, moderate
Convalescence	Relapsing	3rd degree, severe

The *initial stage* is characterised by increased restlessness and perspiration, indicating changes in the child's central nervous system. In this phase symptoms of altered mineral metabolism are likewise manifested by a slight softening of the edges of the skull bones, weakly expressed softening of the occiput (cranio-tabes), small changes in the skeletal bones—rachitic beads (costo-

chondral prominences) on the ribs, and a slight widening of the epiphyses of the long bones.

Duration of the initial stage is one or two months.

The stage of *greatest intensity* is characterised by further changes in the bones and by the formation of more or less pronounced deformities of the skeleton. Changes are also observed in the muscles—atonia and impairment of static functions, connected with disturbances of the function of the central nervous system. During this period an enlargement of the liver and spleen are also observed.

The stage of *convalescence*, of recovery, is characterised by the subsidence of the enumerated symptoms of rickets. The skull bones thicken, craniotabes disappears, the static functions are restored.

Aftereffects are most commonly observed in children over two and three years of age. The rachitic process is over by this time, but skeletal deformations remain, as well as changes in the teeth (thin enamel coatings, early caries). Sometimes the child is anemic during this period.

Notwithstanding the fact that the process is presumably ended, unfavourable conditions (illness, malnutrition) may evoke relapses.

An *acute course* of the disease is characterised by a rapid development of the symptoms of rickets. First and foremost are manifestations of disturbances in the central nervous system (restlessness, perspiration). These symptoms are frequently observed in the first months of life, particularly in premature infants, if measures for preventing rickets have not been taken.

A *subacute course* of rickets is manifested by the slow development of the disease. The first symptoms are the appearance of a rachitic rosary (beads) on the ribs, bosselation on the cranial bones, widening of the epiphyses of the long bones—that is, development of osteoid tissue, and not softening of bony structures. Such a course is most commonly observed in hypotrophic children, and also in cases when the child is affected at a more advanced age—later than 9-12 months.

A *relapsing course* is characterised by periods of improvement (in summer) and aggravation (in winter) of the rachitic process. The possibility of relapses should be considered, and prophylactic measures taken before the winter season.

1st degree is a mild form of rickets characterised by weekly manifested symptoms in the bones and nervous system and not very marked change in the general condition of the child.

2nd degree is a moderately severe form of rickets distinguished by moderately expressed changes in the bones, muscles, nerves and in blood formation and marked impairment of the general condition. In this form of rickets the liver and spleen may become enlarged and a moderate anemia develop.

3rd degree is a severe form of rickets. It is marked by significant changes in the bones (skeletal deformities), muscles (muscular atonia), nervous system (static and motor disorders), and in hemopoiesis (severe anemia). In this form of rickets the liver and spleen are also frequently enlarged.

The above classification is a guide to establishing the form of the disease, although there naturally may exist transitional forms; this must be taken into consideration in determining the most effective method of treatment.

Rickets should be diagnosed as early as possible, while no changes have as yet appeared in the bones. The earliest symptoms of rickets are restlessness, loss of appetite with no visible reason, poor sleep, a pale face, cold, pale extremities that may even have a bluish tinge, the skin loses its velvety softness, profuse perspiration of the back of the head, an ammoniacal odour of the urine. These symptoms are sufficient grounds for diagnosing rickets if all other pathological conditions may be excluded.

Measures taken at this time may prevent the further development of rickets.

Prophylaxis of rickets should be initiated before the birth of the child (antenatal prophylaxis).

Antenatal prophylaxis consists of the following measures: the pregnant woman's food must contain a sufficient amount of minerals, vegetables, fruits, milk and vitamins (A, B₁, C, D); she should be outdoors as much as possible, and during the last 3-4 months of pregnancy should take vitamin D for prophylactic purposes (2,000-3,000 u daily).

For postnatal prophylaxis of rickets housing and domestic conditions must be improved. Thanks to the improvement in housing conditions after the October Revolution and the widespread propaganda of breast-feeding without additional foods until six months of age, and of exposing children as much as possible to fresh air and sunshine, severe forms of rickets have entirely disappeared in our country. A great health-promoting role is played by various child-care centres (crèches, child homes, and infant health centres). The environment in these institutions are favourable, the children are fed and managed properly. Babies should never be given cow's milk in excess; moreover, it should be seen to that

the milk is of *good quality*. At two months, particularly in cases of bottle-fed and premature infants, vitamins C and D should be prescribed in the form of fruit juices and fish-liver oil; the latter must be started with great caution (to avoid diarrhea), beginning with several drops and ending with one teaspoonful twice a day. Fruit juices are permitted in large quantities (up to 50 ml a day in the second half year of life). Vitamin D is also given for prophylactic purposes (3,000-5,000u per day), beginning with the second or third month of life; it is recommended to administer the above dosage of vitamin D for one month, and after a month's interval for another two months, three one-month courses in all (for instance, November, January, and February). Besides keeping the child out-of-doors as much as possible, he should also be exposed to sunshine, only overheating must be avoided. A good preventive measure is irradiation by mercury vapour lamps, a source of short-wave ultraviolet rays.

Treatment. Improvement of housing conditions, regular airing of premises, being constantly out in the open in the summer and 4-6 hours in the winter and autumn months, extensive use of sunshine, cleanliness, good care, proper management of nutrition, breast-feeding—all these are essential factors in the effective treatment of rickets. Great importance is attached to keeping children in the fresh air away from town. An old and tested method is the prescription of cod-liver oil as it contains vitamins A and D (the growth vitamin and the antirickets vitamin). Fish oil is also obtained for medicinal purposes from dolphins, whales, and seals. The oil should be kept in a cool spot; in this case it may be given the year round, even in the summer. It is first given in drops, 5-10 in a little milk, and brought up to two teaspoonfuls a day (given at two feedings) for infants and three teaspoonfuls a day before meals in the second year of life. The majority of babies get used to fish-liver oil and take it readily; however, if the child refuses it, or starts vomiting, the oil should be given in some vehicle, such as kissel (fruit or berry starch pudding). A disadvantage of fish-liver oil is that its vitamin D content is irregular. Vitaminised fish-liver oil with a definite vitamin D content is now available. Egg yolk also contains vitamin D. It is recommended to give young children (in their second year of life) caviar (fish roe), as it also contains vitamin D. For the treatment of rickets a special vitamin D is administered. It is obtained by ultraviolet irradiation of ergosterin (ergosterol) extracted from yeast; an oil solution of radiated ergosterin is on sale under the name of vitamin D (vitamin D₂).

Vitamin D is measured in international units (u). One ml of a vitamin D preparation may contain from 10,000 to 30,000 u (1 ml of vitaminised fish-liver oil contains 250 u of vitamin D).

The therapeutic dose of vitamin D for breast-fed infants with a mild form of rickets is 5,000 u a day. In more severe cases, and also when babies are either on a mixed diet or entirely bottle-fed, the vitamin D dose is raised to 10,000-15,000 u. Duration of treatment is two months, and subsequently the child is given the ordinary kind of fish-liver oil (nonvitaminised) throughout the year.

Prolonged administration of fish-liver oil may lead to secondary effects (hypervitaminosis).

Vitaminised fish-liver oil may be used therapeutically if the precise amount of vitamin D contained in it is computed for a definite course of treatment. The vitamin D preparation is available in solutions of oil or alcohol; the latter contains 200,000 u of vitamin D in 1 ml. It is mostly used in the so-called shock method of treatment, when vitamin D is administered for short periods—600,000 u within three to ten days. In individual cases 1,000,000 u are prescribed for a course of treatment. However, such a method should be applied to hospitalised patients only. In domestic conditions the oil solution alone should be administered, in drops computed according to the vitamin D content in 1 ml as specified on the label.

Significant overdoses of vitamin D preparations, particularly when given for prolonged periods of time, may lead to the appearance of symptoms of hypervitaminosis: loss of appetite, listlessness, drowsiness, elevation of temperature, albumin in the urine, hyaline casts, leukocytes. In such cases the administration of vitamin D should be discontinued immediately and not resumed before at least a month has passed. It is recommended to combine the vitamin D treatment with a simultaneous administration of calcium preparations (10 per cent solution of calcium chloride, calcium lactate and calcium gluconate).

Sunbaths are also employed in the therapy of rickets.

Exposure to *sunshine* (heliotherapy) as a treatment for rickets is conducted after a special technique, beginning with 1-2 minutes and ending with 15-20 minutes, in the morning. The child's general condition must be carefully observed. In the winter the entire body is exposed to artificial ultraviolet radiation as a therapeutic measure, in all 15 to 20 exposures on alternate days. The child's clothing is removed and he is placed for three to fifteen minutes under the lamp, at a distance of 100 cm from it. Carelessness in radiation dosage may evoke redness of the skin (ery-

thema); however, this disappears soon when oil is applied to the red area. Protective glasses must be put over the child's eyes. The hydropathic procedures employed with relative success are saline baths (100 g of table salt to a pail of water) at 34-35°C (a total of ten baths on alternate days), and saline sponge baths of the whole body, limb by limb (one teaspoonful of salt to a glass of water). Gymnastic exercises and massage are most beneficial for infants. As the bones of rachitic children become brittle, such children must not be allowed to sit up too early, or to walk until they become stronger. Babies should be carried on both arms alternately in order to avoid possible spine curvatures. Upon the appearance of deformations the child should be given timely orthopedic (corrective) treatment.

In the therapy of rickets attention must be paid to proper feeding and nutrition. The child should have foods containing other vitamins besides D; this pertains chiefly to vitamin C (raw fruits and their juices, vegetables). A monotonous milk and cereal diet is to be avoided. At 6-7 months it is good to give babies a raw egg yolk in cereal or vegetable purée; rachitic babies may be given egg yolk somewhat earlier (at 5-5½ months).

For proper treatment and control of rickets all rachitic children should be on a special list in the district infant health centre or polyclinic. Sanitary propaganda activities among mothers constitute another very important item.

Spasmophilia

Spasmophilia is a morbid condition characterised by a state of intensified excitability of the neuromuscular system and by a number of symptoms displayed either separately, or together: a) sudden attacks of generalised clonic spasms (eclamptic seizures); b) attacks of spasmodic closure of the glottis (laryngospasm); c) seizures of spastic convulsions of separate groups of muscles (tenany).

Clinical findings. Two forms of spasmophilia are distinguished, a latent and an open one. Latent spasmophilia may be diagnosed by a number of signs. Chvostek's sign: a tapping of the face in front of the ear produces rapid spasms of the upper lip, nostrils and eyelids (excitation of the facial muscle); Trousseau's sign: carpal spasm can be elicited by compressing the upper arm (compression of the neurovascular bundle in the sulcus bicipitalis), all five digits coming together in a characteristic cone-shaped form called accoucheur's or obstetrician's hand (not always observed);

Erb's sign: hyperexcitability is determined by the use of a galvanic current. D.C. below 5 mA shows a sharp increase in electric excitability. The hands of normal children contract under the action of currents no lower than 5 mA (cathode disconnection). This last is the earliest sign of spasmophilia, when the other signs may not have appeared yet.

In the presence of certain conditions favourable to the development of the disease, latent spasmophilia turns into open spasmophilia, with the following clinical pattern:

Stable tonic convulsions appear in the muscles of the hands (obstetrician's hand), of the feet (carpopedal spasms) and of the facial muscles (resembling facial convulsions in tetanus). Such spasms may continue for some time; in some cases the sphincters of the anus and the bladder are involved with resulting retention of urine and feces; on rare occasions the smooth bronchial muscles are involved, and this brings on asphyxial seizures accompanied by paleness and cyanosis, sometimes simulating pneumonia and bronchial asthma (bronchotetany); another rare finding is affection of the cardiac muscle and nerves in the diastolic period.

Spasm of the glottis (laryngospasm) appears upon inspiration during crying fits and undue excitation; it is accompanied by a moaning sound. The closing of the glottis makes the child pale and blue in the face; gradually the spasm passes and the blueness disappears. A severe attack of laryngospasm may bring on unconsciousness and even death.

Clonic spasms in the face and the extremities may last from several minutes to several hours, and may be repeated up to 15 times in 24 hours. The child is unconscious during the attacks, which may repeat themselves on several consecutive days. A new attack of clonic spasms may return in several weeks or months; severe seizures are sometimes fatal.

In obvious spasmophilia the patient may display several of the symptoms, or only one.

Prognosis should be guarded for laryngospasm and general convulsions, as fatal terminations are sometimes observed.

Prophylaxis. Improvement of domestic conditions, breast-feeding, sufficient intake of vitamins (fish-liver oil, fruit juices) and extensive exposure to fresh air and light are a good safeguard against this disease. Particular care should be taken in this respect of rachitic children, premature and bottle-fed infants.

Treatment. In cases of latent spasmophilia the child should be taken outdoors every day for four to eight hours (with intervals), he should be given vitamin D, or vitaminised fish-liver oil, and

cow's milk should be excluded altogether for 2-3 weeks. If breast milk is not available, the baby is given kefir and acidified mixtures; after 12 months milk may be excluded for a time and replaced by curds (cottage cheese) and kefir (10-30 g in cereal). Calcium chloride is given (1-2 g per day) in 10 per cent solutions.

In obvious spasmophilia accompanied by laryngospasm the child should not be exposed to the sun for too long, he should be protected against all manner of excitation, bruises, infection. During severe seizures of laryngospasm accompanied by unconsciousness the child should be carried to an open window, his tongue drawn out, and artificial respiration applied. For generalised clonic spasms chloral hydrate enemas are given (2-3 tablespoonfuls of a 2 per cent solution to an enema) and the room is thoroughly ventilated. The child must be taken outdoors every day for a total of six to eight hours; he must be given fish-liver oil or vitamin D and calcium chloride, and no cow's milk at all for two to three weeks. For sleeplessness and restlessness sedatives are indicated—calcium bromide and luminal. The employment of ultraviolet radiation and large doses of vitamin D is not recommended for such patients. Tonic convulsions are treated in the same manner as latent spasmophilia.

PATHOLOGIC CONDITIONS DUE TO VITAMIN K DEFICIENCIES

Vitamin K (antihemorrhagic factor) occurs widely in nature. It is found in the tissues of many plants, such as carrot greens, spinach, cabbage, pumpkin, cauliflower, nettles and others, and also in pork liver and fish-meal. However, its content is highest in spinach, pumpkin and cauliflower. Vitamin K is also formed in the human intestine by many bacteria (*Escherichia coli* and others).

Natural vitamin K is oil-soluble. In the intestines where it is formed and also delivered with food it is absorbed together with the fats. Soviet scientists have evolved a water-soluble preparation of vitamin K called vikasol.

Vitamin K is essential in the process of formation of prothrombin and it is therefore an important factor in blood clotting. The deficiency of this vitamin in the body leads to prolongation of blood-clotting time and to the appearance of hemorrhages in the skin, mucous membranes and various internal organs.

Several forms of vitamin K deficiency are encountered in children.

Melena. *Melena neonatorum* is an extravasation of blood into the stomach and intestines of the newborn infant; this disease is characterised by blood-coloured stools and vomitus, and by nasal and umbilical hemorrhages. During postmortem examinations gastric and intestinal ulcers are sometimes found. The disease is manifested on the second or third day of life and its mortality rate in severe cases is high.

As investigations have shown, this disease is caused by a sharp decrease of the *prothrombin* level in the blood of the newborn from the second to fifth day after birth. Vitamin K facilitates the formation of prothrombin, but the newborn have almost none of it, as this vitamin does not pass through the placenta easily, while the intestinal flora, the source of vitamin K in the body, has not appeared yet. In these cases melena is successfully treated with 10 ml intramuscular injections of maternal blood, placental blood, measles convalescent serum or diphtheria antitoxin, and also the transfusion of citrated blood into a vein of the scalp (5 ml per kg of weight), as the blood of adults has a high prothrombin level. Of late vitamin K has been given orally for raising the prothrombin level, in 5 mg doses twice a day with 0.1 g of sugar (in milk). The effect of the introduction of vitamin K becomes apparent in 4-6-8 hours, the bloody stools and vomitus soon disappear. If hemorrhages do not stop another similar dose of vitamin K is given in 12 to 14 hours, but no more than on three consecutive days. The same pertains to blood injections if vitamin K is not available. Melena appearing after the fifth day of life is usually a result of umbilical sepsis and often has a fatal termination.

Hemorrhages under the dura mater. Besides conspicuous birth injuries such as rupture of the sinus of the dura and of the lateral lobes of the cerebellum, there may appear after the second day of life spasms, cyanosis, skin hemorrhages, tension of the fontanelles, at times even pareses of the extremities, and on subsequent days an elevation of temperature to 38°. All this is the result of prothrombin deficiency in the blood (hypoprothrombinemia) and of hemorrhaging into the dura mater of the brain and spinal cord. These symptoms sometimes intensify and the child dies.

Hemorrhages of this type are particularly frequent among premature infants. Even in cases of recovery certain changes of the central nervous system are retained in some children.

Treatment, similar to that employed for melena, is effective on the first day after the onset of the disease.

PATHOLOGIC CONDITIONS DUE TO DEFICIENCIES OF THE VITAMIN B COMPLEX

The vitamin B complex is an association of a still unknown number of vitamins contained chiefly in the integuments (seed-coats) of various grains, in the liver, and in particular in baker's and brewer's yeast. In childhood pathology diseases connected with vitamin B₁ and B₂ deficiencies are the most important.

Vitamin B₁ Deficiency

The disease caused by lack of vitamin B₁ was first described in 1888 under the name of beriberi. It is seen in countries where the population lives mainly on a polished rice diet (China, Japan, India, Indonesia, the Philippine Islands), as the vitamin is contained in the grain hulls. It usually affects adults and older children, but 2-3 months old breast-fed infants may also get it if their mothers are ill. Infants reared on a mixed diet, or entirely bottle-fed, are rarely affected. The disease manifests itself in the following forms:

the *cardiovascular* form, accompanied by hypertrophy of the right ventricle, an accentuated second sound and an accelerated pulse followed by a galloping rhythm and dulled sounds;

the *nonprotein edematous* form;

the *neuritic* form, accompanied by *paralyses*, lesions of the sensory and motor mechanisms, dizziness, headache, spasms, and rigidity of the neck.

These forms of beriberi are often interlaced, converting from one into another; they are the different stages of one and the same disease.

The cardiac form terminates lethally within two days. The duration of beriberi in children is two days to two weeks with a general mortality rate of 30 per cent, while the mortality rate of the cardiac form is 95 per cent. A sharp right ventricular hypertrophy is observed in beriberi patients.

B₁ hypovitaminosis in children often appears in association with other diseases, particularly with prolonged gastrointestinal illnesses. Its beginning is usually connected with a loss of appetite (anorexia) caused by the basic disease. If this goes on for a long time, the child does not ingest the necessary amount of vitamins (including vitamin B₁). A very important factor in the development of B₁ hypovitaminosis is a disruption of the normal formation of this vitamin by the intestinal bacteria as a result of pro-



Fig. 22. Weeping Eczema on Forehead and Cheeks



Fig. 23. Dry (Seborrheic) Eczema

tracted digestive disorders. Moreover, doctors often prescribe carbohydrate diets for digestive disorders; these diets consist of various cereal waters, liquid creamed cereals and gruels with rice and refined wheat (farina), white bread and toast. Such food contains no vitamin B₁ and promote a progressive development of the hypovitaminosis. The condition can also develop in breast-fed infants if their mothers' diet lacks vitamin B₁. The first symptoms of the disease are usually paleness, loss of appetite, constipation, failure to gain weight, the appearance of an accentuated second sound in the pulmonary artery. Later the heart sounds become dulled, severe emaciation occurs, muscular hypertonia, rigidity of the neck, and spasms resembling tetany (spasmophilia) appear. In a number of cases the feet swell up (edema).

Such symptoms were observed in children formerly, too, but an incorrect explanation was offered for them—they were looked upon as simple cases of hypotrophy. Usually such children do not gain in weight without vitamin B₁, and their neurotic symptoms increase. Immediate results—weight gains and decrease of neurotic symptoms—are attained by including kefir in the child's diet and giving him injections of vitamin B₁ (thiamine) subcutaneously for 10-15 days, in 0.5-1 ml doses.

Good results are obtained by adding baker's yeast to the baby's cereal (one-fourth to one teaspoonful per cup of cereal); the cereal is brought to a boil after the addition of the yeast (after G. Spersky).

Vitamin B₂ Deficiency

Vitamin B₂ is a complex of vitamins of which more than ten are at present known. The ones best known in their relations to the human organism are: a) *riboflavin* (vitamin B₂) and b) *niacin* or nicotinic acid (the PP factor or vitamin).

a) *Riboflavin* occurs abundantly in nature, most of all in yeast, liver, kidneys. Similar to vitamin B₁, it is synthesised by the intestinal flora. The lack of this substance in the bodies of children and adults causes inflammatory processes on the mucous membrane of the tongue (glossitis). The tongue becomes red, its mucosa dries, cracks, macerates; fissures appear in the corners of the mouth (stomatitis angularis); redness and maceration of the lips, of the wings of the nose, over the eyebrows, on the ears (seborrheic stomatitis and dermatitis) develop; the conjunctiva of the eye becomes hyperemic (bloodshot). In order to prevent the development of riboflavin deficiency the child's diet should be corrected to include a sufficient amount of meat, eggs, milk.

Spinach, peas, and from time to time liver and kidney are also highly recommended.

Besides the enumerated dietary measures, therapeutic doses of pure vitamin B₂ (riboflavin) are prescribed: for very young children 5-10 mg per day, for older children 15-20 mg, the total daily dose being divided into four or five smaller doses. Complete relief is obtained by this treatment within 2-3 weeks.

b) *Niacin* (nicotinic acid) also occurs widely in nature in products of plant and animal origin. Its greatest dietary source is mushrooms, yeast, meat, liver, and kidneys.

The lack of nicotinic acid in the body may be due to certain pathologic conditions, as, for instance, anemia. Anemia is accompanied by a lowered acidity (hydrochloric acid) in the gastric juice and protracted intestinal disorders associated with failure of absorption of many vitamins, including niacin (endogenic deficiency).

The most marked manifestation of niacin deficiency occurs in its complete absence, or avitaminosis. This disease is called *pellagra*, and hence its other name, vitamin PP (from the words "pellagra preventive"). Pellagra is a disease of all ages. It is observed in southern countries where the principal food of the population is cornbread and other maize products, and no meat or milk at all are consumed. In the clinical aspect of pellagra the most characteristic symptoms are pigmentation of the open areas of skin with subsequent scaling, diarrhea, lesions of the oral mucosa (glossitis), neuritis (general depression, apathy, absence of all interest in surroundings, and in protracted cases severe emaciation).

Pure pellagra is very rarely observed in the Soviet Union. However, children may manifest mild symptoms of vitamin PP deficiencies, each of the above-described symptoms being very light, their combinations unclear, and some of them altogether absent. Usually such deficiencies develop in association with very protracted intestinal disorders or some other emaciating diseases resulting in severe dystrophy that evokes some of the symptoms of pellagra (a so-called pellagroid condition). Intestinal and nutritional disorders of this type in children do not respond to usual methods of treatment. In some of these cases a slight pigmentation is noticed on the forehead, neck, back of the fingers, on the thighs and shoulders. It looks as if the skin were dirty, or, in some instances, as if the patient were dark-skinned or sun-tanned. A child so affected takes hardly any interest in toys, answers questions unwillingly.

Treatment consists of providing a suitable nutrition of full dietary value, and of prescribing pure nicotinic acid. The total daily doses of vitamin PP for different age groups are: for children younger than 3-4 years 10 to 15 mg in 2-3 portions; for preschool children 20 to 30 mg; for children of school age 0.05 to 0.1 g. In the presence of diarrhea it is better to introduce the preparation by intramuscular injection of a 1 per cent solution of niacin in amounts varying from 1 to 10 ml, depending on the patient's age.

In some children the introduction of niacin, both oral and intramuscular, evokes redness of the face, ears, neck and front of the chest, a burning in the mouth and a sensation of insects creeping all over the body. Usually this vascular reaction disappears in 20 to 30 minutes.

PATHOLOGIC CONDITIONS DUE TO VITAMIN C DEFICIENCIES

Vitamin C is a substance occurring chiefly in the green parts of plants and in edible roots, in fruits and berries; its greatest dietary source is black currants, dogrose hips, lemons, oranges.

Science has made clear the nature of vitamin C and it is now produced synthetically under the name of ascorbic acid.

Complete absence, or even deficiency of vitamin C in the food causes a severe disease known as scurvy.

The disease is now observed very rarely, even in conditions conducive to it (protracted sea journeys and military marches), in connection with the widespread consumption of natural products containing vitamin C, and of its synthetic analog ascorbic acid. Formerly infants were often apt to have scurvy when, in the absence of breast milk, they lived on various cow's milk substitutes (sterilised cow's milk, various milk concentrates, children's flour preparations).

Clinically scurvy in children was characterised by a peculiar condition of the bones. First they became tender to the touch, then swelled, most commonly in the femoral epiphyses, further subperiosteal hemorrhages occurred, the entire thigh swelling and becoming very painful. Hemorrhages also appeared beneath the periosteums of the ribs (marked beading resembling a rachitic rosary). Small and large hemorrhages occurred into the skin. Urinalysis often showed red blood cells, and sometimes the stools contained blood. Babies were more commonly affected in the second half year of life; sometimes the illness dragged on for months, and often various complications were superimposed on

it, and in cases where the disease was not diagnosed in time and where no proper treatment was instituted the termination was fatal.

Although we do not at present observe such severe displays of vitamin C deficiency in children, weaker forms of scurvy, called hypovitaminosis or vitamin C deficiency, are still observed.

People of any age are apt to be affected by vitamin C deficiencies during certain seasons (mostly the winter and spring) when food does not contain enough of it and its level in the blood goes down sharply. A similar decrease of the vitamin C level in the child's body may take place in the course of certain illnesses.

The symptoms of vitamin C deficiency do not arise spontaneously. If the disease develops in an obscure form weeks or even months may pass before the symptoms become evident. The earliest sign of vitamin C deficiency is fatigability, loss of appetite, drowsiness. Later the gums start bleeding. Hard crying or coughing evoke pin-point hemorrhages on the child's skin (the neck, eyelids, elbow bends).

Urinalysis shows a small number of red blood cells, and the child's temperature may be slightly elevated at times.

This condition is commonly of an undulating nature. The process is protracted, showing improvement in the summer and autumn months and aggravation in the winter and spring. During aggravation of the condition children sometimes complain of pains in their legs and feet and stop walking.

Treatment. First of all it is necessary to ascertain the cause of the illness. If it is only insufficient intake of vitamin C with food, then fruit and berry juices, dogrose hip decoctions, or ascorbic acid, are prescribed. The presence of another disease that may be conducive to the vitamin C deficiency requires corresponding treatment and an additional amount of vitamin C, no matter what quality of food the child is being given. The daily dose of vitamin C is 150-200 mg of ascorbic acid divided into three or four smaller doses.

Improvement is noticeable after a week or two of treatment; however, complete recovery occurs much later. Therefore, vitamin C deficiencies in children should be treated for one and a half to two months.

It has been noticed that when scorbutic children are given pure ascorbic acid the effect of this treatment on the hemorrhages is not as good as that obtained with natural antiscorbutic products. Best results are obtained with lemon juice. A close study of natural vitamins has shown that dietary products such as lemons,

red pepper and some others contain, besides ascorbic acid, a factor that supports capillary impermeability in the vessels, and in cases when permeability is increased, as in scurvy, it strengthens the capillaries. This substance has been isolated in its pure form from lemon, red pepper, grapes, plums and other products and called *vitamin P* or *citrin*. In many fruits and berries citrin is always associated with vitamin C in varying ratios.

EXUDATIVE DIATHESIS

Exudative diathesis is a peculiar tendency of the skin and mucous membranes to inflammatory catarrhal (exudative) processes associated with lesions of the neurotrophic functions of the central nervous system.

Children who have exudative diathesis differ from healthy children by a frequent exhibition of a vivid reaction to slight external irritations of the skin and mucosa (hypersusceptibility). In these children inflammatory catarrhs involving the skin and mucous membranes persist much longer than in children who have no such tendency. This tendency of the tissues to display all manner of exudative processes may evidently become a fixed symptom that is passed on genetically, but one that may be alleviated significantly and even altogether eliminated by various measures—alteration of dietary habits, care, training.

Children affected by exudative diathesis are allergic* (they possess a peculiarly intensified reaction capacity) to certain substances that are chiefly contained in milk, eggs, cocoa, chocolate, less frequently in honey, flour and butter. These products, particularly if given in excess, are *allergens* for these children. Allergens are substances that evoke allergic reactions of various degrees. Different children react to different allergens. Thus, in some individuals exudative symptoms are evoked by eggs or milk, while in others it is sometimes difficult to establish just what foods cause the given symptoms.

Exudative diathesis commonly appears in infancy, in some cases as early as the first months of life.

* The word "allergy" comes from the Greek word "allos", meaning "strange", or "alien", and "ergia", meaning action. So allergy means a strange, or alien action (intensified reaction) as compared with the usual one; in other words, the reactivity of allergic children differs from normal reactivity (or susceptibility) to the introduction of certain substances, chiefly of alien proteins.

Exudative manifestations may also be observed in children older than a year. The various clinical findings in exudative diathesis depend on the child's age.

The exact nature of this pathologic condition in children has still to be clarified. Evidently, it is associated with a significant alteration in metabolism, chiefly fat and water metabolism. There can be no doubt that the disease is connected with lesions of the normal functions of the central nervous system as the principal regulator of all vital activity, including metabolic processes.

Besides alterations in the skin and mucous membranes observed in exudative diathesis, changes are also often noted in the lymphatics (enlargement of local groups of lymph nodes).

The manifestations of exudative diathesis vary depending on the age of the child, his individual traits, and the care that is taken of him. Two types of children affected by exudative diathesis are distinguished: a pastose type and an erethitic type.

Children of the first, *pastose*, type are those in whom all skin responses are characterised by ordinary exudation. They are often afflicted by wet or weeping eczema, are usually stout and not very active. This type of exudative diathesis is usually observed in children who are given too much cow's milk.

The *erethitic* type includes children who have no abundant wet exudations on the skin, but only dry eruptions. Such children, notwithstanding correct management of breast-feeding, usually fail to gain well in weight and they often pass watery green stools.

Clinical findings and forms of the disease. Skin eruptions. "Gneiss"—the appearance over the eyebrows or on the forehead of grey/or yellow scales that later spread over the entire scalp, resembling seborrheic dermatitis (eczema seborrheicum); the scalp is covered with thick crusts that reveal red inflamed patches of skin when removed. "Milk crust"—redness and scaliness of the skin on the cheeks accompanied by a severe itch; the skin is often scratched and the abrasions frequently form purulent crusts connected with secondary infections; subsequently the process may involve the entire surface of the face. *Weeping (or wet) eczema* on the cheeks and scalp—the skin is red and moist (Fig. 22). *Erythema*—redness of the skin in the groin, on the neck, in the armpits; notwithstanding scrupulous care, this condition may gradually turn into wet eczema. *Seborrheic eczema* mostly involves the hairy area of the scalp, covering it with thick crusts; upon their removal the skin underneath appears moist and bleeding. Abundant scaliness is often observed on the body. A dry seborrheic eczema may also erupt on the face (Fig. 23). *Nettle rash* or

hives (urticaria) is characterised by pale pink, intensely itchy wheals or vesicles of irregular shape, markedly elevated above the surface of the skin, ranging from the size of a small pea to a small coin; some of the welts resemble the marks left by stinging nettles. *Strophulus* (also called red gum) is a skin condition characterised by small red vesicles (the size of a lentil seed) that appear on the trunk and limbs; then these vesicles become purulent and extremely itching, and a few days later they dry up. *Prurigo* is an itching nodular rash on the flexor surfaces of the extremities that does not let the child sleep at night; scratching opens the way for secondary infections and purulent vesicles appear on the scratched parts.

Nettle rash, strophulus and prurigo most commonly affect children older than one year, while the skin lesions described lower are observed almost exclusively in infants.

Lesions of the mucous membranes.

Geographic tongue (Fig. 24) is a localised thickening of the epithelium of the tongue



Fig. 24. Geographic tongue

(whitish patches and scalliness), giving the surface the appearance of a map; this condition is very persistent and does not clear up for a long time. *Catarrhal conditions in the throat* (sore throat) and in the *intestines* (dyspepsia). *Conjunctivitis.*

Lesions of the respiratory system.

Laryngitis, protracted bronchitis; bronchial asthma is no uncommon occurrence in children of a more advanced age subject to exudative diathesis.

The *lymphatic glands* (neck, axilla, and groin) are more or less enlarged and hard.

Water metabolism disturbances.

Children who have exudative diathesis do not gain weight normally; their weight gains go up and down owing to the abnormal retention and elimination of water. Water losses associated with various illnesses make these children lose weight very rapidly (100-200 g a day).

Lowered immunity in exudative diathesis causes such children

to become easy victims of many diseases, and the disease itself to run a severe course.

Changes in the blood. *Eosinophilia* (5-10 per cent) is often observed in children with exudative diathesis.

Changes in the nervous system. Sleep is often troubled, and at times intense excitability is observed.

Course. The disease is a protracted one, ending in the majority of cases by the age of two or three years. A chronic dry eczema may appear in older children. In the summer the condition is significantly improved.

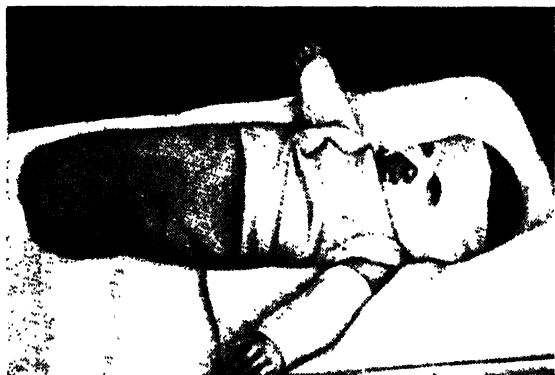


Fig. 25. Gauze mask over face and splints on arms

Prognosis is good.

Prophylaxis consists of proper nutrition and feeding, avoidance of overfeeding (particularly with milk), and of extensive utilisation of sun and air.

Treatment. Restriction of milk and of fatty mixtures for stout children, introduction of vitamins and food of full dietary value for thin babies, the exclusion of eggs and sweets. However, positive results are not always attained even when diet is strictly observed.

The child must never be overclothed nor must he wear tight clothes or be allowed to lie wet. The scrupulous cleanliness of the baby's clothing and of the hands of mother or nurse are essential for the prevention of a secondary infection. A good effect is obtained by air baths given several times a day.

To prevent the baby from scratching the sleeves of his kimona should be sewed up fast, and his arms secured by cardboard splints, so that he cannot bend his arms to scratch his face. In

very severe cases the hands are tied to the sides of the crib by strips of gauze. A gauze mask with holes for the eyes, nose and mouth is placed over the face (Fig. 25).

A good effect is obtained by placing the child in a hammock made of a sheet tied to the walls of the crib at a height of 30-40 cm above the mattress; this provides the entire surface of the patient's body with a current of fresh air.

Diaper dermatitis (diaper rash or intertrigo) is treated by washing the diaper area with a potassium permanganate solution and applying boiled vegetable oil (sunflower seed, cotton-seed, olive oil) to it, or a 2 per cent emulsion of streptocide in fish-liver oil; good results are obtained with starch lotions, and by exposing the affected area to the air for several hours a day.

Hyperemic skin conditions are treated by applications of fish-liver oil, bismuth ointment or paste; for seborrhea of the scalp an oil compress is applied for 12 hours: the crusts are then easily removed, and a 5 per cent solution of silver nitrate is applied to the bleeding, moist skin, followed by fish-liver oil or sulfur ointment. In cases of persistent eczema attended by extensive dry crusting a naphthalane-containing ointment is used. Thin children show improvement when fish-liver oil is given orally. Exudative conditions are also effectively treated by applications of fish-liver oil to the patient's body, face and head. Borax solution compresses are recommended for wet eczema, or a 5 per cent solution of silver nitrate to the skin on alternate days. Extreme itching may be alleviated by adding 2 per cent anesthesin (benzocaine) to the bismuth paste. If the baby cannot sleep because of the itch a 4 per cent solution of sodium bromide is prescribed—one teaspoonful two or three times a day. A rapid disappearance of the severe exudative symptoms is often effected by good care alone. Constipation must be avoided by all means. In some cases the oral administration of 0.01 ml of nicotinic acid three times a day for two to three weeks has promoted a significant improvement in exudative diathesis.

Good reports have been published on the results of tissue therapy in the form of intramuscular injections of aloe extracts (daily doses of 1 ml for 2-3 weeks).

DISEASES OF THE SKIN

The development of skin conditions in children is due to local lesions and to the penetration of infections, as well as to generalised diseases. Many infectious diseases of childhood (measles,

scarlet fever, German measles, meningococcal infections) produce specific lesions of the skin in the form of various types of eruptions. Significant lesions of the skin cause exudative diathesis.

Lower a description is presented of the most characteristic and commonly observed skin diseases in childhood.

PEMPHIGUS NEONATORUM

Pemphigus neonatorum (or it is also called *impetigo* of the newborn) is a disease that affects the skin of infants in the first days or weeks of life. It is characterised by small round reddish spots, the size of a pinhead to a pea that crop out on the body and extremities. Soon these spots turn into vesicles or blebs containing a transparent fluid in which streptococci are found; the fluid becomes clouded and purulent later on, and it then contains only staphylococci, the result of secondary infection.

The blebs are distributed all over the body, except palms and soles (this latter is a feature distinguishing the disease from *impetigo* associated with congenital syphilis). Subsequently the blebs and vesicles rupture spontaneously and dry up, leaving no scar; the smallest lesions may dry up without even rupturing.

The disease is often accompanied by an elevation of temperature (37.5-39°). The course and duration of the disease depend on the dimensions of the affected areas and on the virulence of the infection.

Etiology. Infection probably occurs in the maternity hospital during delivery via the vaginal tract of the mother, the air, or the hands of the midwife or nurse. The primary pathogen is the streptococcus, and secondary infection with staphylococci follows.

Course and prognosis. In some cases the course of the disease is severe, resembling septic pyemia: the skin lesions may develop into phlegmons, abscesses, or be the source of metastatic infections into other organs. Lethal outcomes are possible.

Prophylaxis. Upon the appearance of *pemphigus neonatorum* in maternity hospitals the patients must immediately be isolated and assigned to the care of special personnel. Scrupulous cleanliness of the hands of the staff and of the child's clothing, bedding and diapers is of utmost importance.

Treatment. The most essential thing is good care of the patients and proper feeding. The lesions are treated with solutions of brilliant green, methylene blue, or gentian violet (a 1 per cent solu-

tion in 70° alcohol). Besides this 10 per cent dermatol or xeroform ointment are applied to ruptured lesions, and the infant is bathed in solutions of potassium permanganate (0.2 g to a pail of water) or of tannin (a tablespoonful added to the bath-water). The infant is also injected 2-3 times a day with penicillin, the total daily dose being 10,000 to 15,000 u per kg of weight. Streptocide is also administered, orally: 0.2 g/kg a day in 3-4 doses. In protracted cases penicillin injections are combined with hemotherapy: 10 ml injections of blood are given every day or on alternate days, intramuscularly, a total of 10 to 15 injections; or else 30-40 ml of blood is transfused intravenously over intervals of 5-6 days (4-5 transfusions in all).

EXFOLIATIVE DERMATITIS

Exfoliative dermatitis (dermatitis exfoliativa neonatorum) is a disease affecting the newborn in the first three weeks of life. It is characterised by inflammation of the skin, extreme redness of the face around mouth and nose, and later of the body and lower extremities. Multiple vesicles soon crop out on the red patches; the vesicles rupture and large areas of skin turn into raw red surfaces, resembling second degree burns.

An elevation of temperature is usually observed.

The *etiology* of this disease is not clear. Some authors consider the pathogens to be pyogenic agents (streptococci and staphylococci), while others assume that this violent skin reaction accompanied by shedding of the epidermis is caused by the sudden absorption of certain toxic substances (intoxication), attended by a subsequent permeation of a secondary streptococcal infection. At the height of the disease, when large vesicles or bullae are present, exfoliative dermatitis is an acute streptodermatitis of the newborn, its process outwardly resembling impetigo (pemphigus).

The disease runs an extremely violent course. In most cases it terminates fatally within four to seven days as the result of septicemia or pneumonia.

Prophylaxis. As for other purulent lesions of the skin (impetigo and similar diseases).

Treatment. As for impetigo; penicillin as indicated above, hemotherapy and blood transfusions, local applications of fish-liver oil to the skin, daily baths in solutions of potassium permanganate.

ERYTHRODERMA DESQUAMATIVA (LEINER'S DISEASE)

Desquamative erythroderma is a disease of early infancy (the first three months of life), observed mostly in breast-fed infants. It is characterised by a generalised redness of the skin, beginning with the head and spreading to the body (although in some cases the redness first appears on the buttocks, resembling diaper rash due to bad care). Soon a scaly eruption appears on the reddened surface of the skin, and these scales are shed freely. The scales are greyish-white, greasy, and slightly shiny; they are easily shed and just as easily formed in the same spots. The lesion covering the hairy surface of the scalp resembles seborrheic eczema.

The *etiology* of the disease is not quite clear. It has been assumed that it is associated with systemic intoxication caused by metabolic products; another contributing factor may be the deficiency in breast milk of the so-called skin vitamin (vitamin H, one of the vitamins of the vitamin B complex).

Clinical findings. Erythroderma differs greatly from the dermatitis described above; however, as there are transitory forms ranging from typical dermatitis to erythroderma, some authors (M. Skvortsov) consider that there is a close relation between both of these diseases, and that they are both reactions to one and the same harmful agent or agents. During the first days and weeks of life this reaction produces the violent course of exfoliative dermatitis, while in infants older than three weeks the same process runs a milder and slower course, producing the clinical pattern of desquamative erythroderma.

Histological examinations of the skin show that both reactions are of an essentially similar nature, their difference lying only in degree: one reaction seems to be the beginning of a violent process, while the second reaction is but a slower continuation of this process.

Dyspeptic symptoms are a typical clinical finding in erythroderma; these symptoms often persist throughout the disease, notwithstanding proper management of breast-feeding. All this points to a severe generalised process in the child's organism, manifested by a peculiar skin eruption. Scalp lesions in erythroderma are difficult to distinguish from the seborrheic eczema of exudative diathesis, a condition that may in certain cases spread from the head to the body. However, a characteristic feature of erythroderma is the acute redness of the skin and the continuous shedding of the epidermis in small scales; in erythroderma the skin, when

cleared of scales, never reveals the moistness that is often evident in eczema. Moreover, enlargement of the regional lymph nodes is rare in erythroderma, while it is constantly observed in exudative diathesis.

Prognosis is more favourable for erythroderma than for exfoliative dermatitis; the greater the surface of skin involved, and the more violent and prolonged the dyspeptic displays—the graver the prognosis.

Treatment. In view of the fact that erythroderma in infants is connected with a deficiency of certain vitamins in the mother's milk it is recommended to replace half of the milk the infant gets from his mother by milk from another woman whose child is healthy; if this is not feasible, milk should be obtained from the milk bank at the infant health centre, and if this, too, is not possible, then two or three breast feedings are replaced by protein milk or B-kefir. Local treatments include baths with potassium permanganate, and fish-liver oil applications to the skin several times a day, as for dermatitis. Ultraviolet radiation has a good effect. Thorough cleanliness and good care are essential. In protracted severe cases hemotherapy or blood transfusions are indicated.

In recent years erythroderma has been successfully treated with *campolon*, a liver preparation containing vitamins of the B complex. *Campolon* is given subcutaneously or intramuscularly, 0.5 to 1 ml on alternate days or every day, six to ten injections in all.

Complications (pneumonia, secondary pyogenic lesions of the skin) are treated with antibiotics (penicillin, biomyacin) in doses prescribed for infants.

IMPETIGO CONTAGIOSA

The basic element of impetigo contagiosa is a vesicle or bleb with a very thin, easily ruptured surface; its fluid content is at first clear but rapidly (within several hours) becomes cloudy as the result of secondary staphylococcal infection. The blebs rupture and a thin orange crust forms; the crusts slough off, leaving pinkish spots. The most common site of the lesions is the face. The disease affects not only weak children, but also perfectly strong and healthy ones. It spreads among children in child-care institutions where nursing management is negligent and the premises are not kept in a proper sanitary condition. The course of

the disease progresses without any unpleasant subjective sensations or elevations of temperature. Sometimes the contents of the vesicles dry up into thicker straw-coloured crusts owing to secondary infection, the skin under them becomes moist and eroded; the disease takes on a more protracted character and is accompanied by a swelling of the lymph nodes and may spread over large surfaces of the body.

Treatment. The patient must not touch the involved surfaces to avoid spreading the infection to healthy areas of the skin. The drying crusts are brushed with brilliant green or methylene blue. Good results are also obtained with white precipitate ointment (ung. praecipitatum album).

BULLOUS IMPETIGO

Bullous impetigo or impetigo bullosa. This form of impetigo is characterised by bullae, or large blisters, often the size of a hazelnut. These blisters are sometimes dispersed all over the body, often on the legs and around the nails. While the fluid content of the bullae is clear it contains a pure streptococcal culture; this has given rise to another name of the disease: bullous streptoderma. This form of impetigo is more serious than the ordinary form, and the patient's temperature is often elevated. After the crusts fall off a pink scar remains on the skin, but in cases of a massive secondary staphylococcal infection, as can also happen in ordinary impetigo, ulcerations may form under the crust.

Prognosis is good in most cases; however, certain cases of bullous streptoderma may run a very severe course, leading to the development of clinical symptoms of septicemia with metastases in other sites.

Treatment. Local treatment is similar to the treatment employed for ordinary impetigo, except that penicillin is indicated from the very onset, as there can be no assurance that bullous impetigo will not be complicated by septicemia. In severe cases hemotherapy and blood transfusions are required.

MULTIPLE SKIN ABSCESESSES, OR PYODERMA

Pyoderma or multiple abscesses of the skin (Fig. 26) is characterised by the appearance of multiple lesions on the trunk, head and extremities; the lesions are reddish pus-filled swellings rang-

ing in size from a pea to a hazel-nut. This type of pyodermatitis most often affects feeble babies, particularly when sanitary conditions are bad. Pyoderma may also affect healthy children if the people who care for them have purulent skin lesions on their hands. Pyoderma may appear at the end of severe illnesses (dysentery, scarlet fever, measles, pneumonia). The multiple abscesses are an inflammatory reaction of the epiderm to the penetration of staphylococci into the efferent ducts (outlets) of the sudoriferous (sweat) glands. In distinction from furunculosis pyoderma is a lesion of the sweat glands, while furunculosis affects the sebaceous glands. In furunculosis there is always a firm infiltrate around the sebaceous gland that does not soften for a long time; in pyoderma the infiltrate rapidly softens and disappears, while the surrounding dermal tissue and glandular elements in it become necrotised, forming subdermal ulcers that may remain soft for weeks; when these ulcers are lanced they never produce a thick core, as in furunculosis. The clinical findings include, besides local symptoms, an elevation of the patient's temperature ($37-38^{\circ}$ and even higher). The disease may persist for months, often emaciating the child and terminating fatally owing to pneumonia as a secondary affection.

Prophylaxis. Thorough cleanliness of the baby's clothing and bedding are absolutely essential, and the hands of the persons caring for the child must be thoroughly washed every time before the child is handled. Persons with pyogenic infections on their hands must not be permitted to touch infants.

Treatment. First of all the child's lowered resistance to disease must be restored to normal by the institution of a proper dietary regimen. Infants must be provided with breast milk. Vitamins A and C are necessary for children of all ages; patients should be taken out into the fresh air for as long as possible. In severe cases blood transfusions are indicated. Multiple abscesses, particularly in infants, sometimes lead to generalised toxemia and pyemia; in these cases penicillin therapy is necessary. A good local effect is obtained by hot baths ($39-40^{\circ}\text{C}$) with potassium permanganate, for five to ten minutes.



Fig. 26. Multiple skin abscesses

Part of the lesions rupture in the bath, part of them resolve. The condition responds at times to ultraviolet radiation.

Albucid is administered orally (0.3-0.5 g four times a day).

MILIARIA (PRICKLY HEAT)

The secretion of sweat in the first month of life is not sufficiently developed; in subsequent months perspiration appears, and may even become abundant, particularly in rachitic babies. Such an excessive secretion of sweat may lead to certain pathologic conditions.

Miliaria (or prickly heat) may appear as an irritation of the skin around the openings of the sweat glands even in early infancy; it is commonly observed when the infant's skin is not properly looked after, particularly when rubber or oilskin pads are wrapped around the baby together with his diapers or when his wet diapers are not changed often enough. In such cases a fine reddish rash, resembling that of scarlet fever, appears on the skin, most commonly on the chest, back, and face. Its termination is usually benign, the rash disappearing as soon as the causative factors are eliminated (overheating, excessive clothing). However, in some cases, when the infant is not taken proper care of, a secondary pyogenic infection may penetrate the lesions; in such cases the small reddish spot (the primary element of miliaria) may become the site of an accumulation of pus, forming into a purulent vesicle containing staphylococci. This pustule or vesicle may subsequently serve as the primary seat for pyoderma and other pyogenic lesions of the skin.

Ordinary miliaria (miliaria crystallina or sudamen) may affect children of a more advanced age, as well as adults. The basic element of this skin condition is a small, clear, transparent vesicle containing sweat; the vesicles appear on the trunk or flexor surfaces of the extremities, often in crops. The appearance of such vesicles in children is the result of excessive perspiration, a condition that often accompanies certain infectious diseases, and also when the child is dressed too warmly. The vesicles never become purulent, and they dry up within several days, forming small scales. Such accumulations of sweat under the epidermis may cause the temperature to rise and aggravate the child's general condition.

Prophylaxis and treatment of miliaria consist of keeping the child's body clean, permitting no overheating or excessive cloth-

ing, exposing his body to the air (air baths). In severe cases, particularly if miliaria develops into pyoderma, baths with potassium permanganate are beneficial.

RINGWORM

Ringworm or trichophytosis is a contagious disease of the skin, hair and nails caused by the invasion of the *Trichophyton* fungus. The lesions caused by this fungus are called tinea. The clinical aspect of the disease differs with localisation of the lesions.

Body ringworm or tinea corporis is an infection involving the nonhairy (glabrous) skin. Slightly raised circular reddish spots differing sharply from the surrounding skin appear; these spots rapidly turn into large patches. A reverse process soon commences in the middle of the patches: the redness fades, a depression forms and the skin in this area is covered with mealy scales. The lesions are most commonly seen on the face, neck, and arms.

Ringworm of the scalp (tinea capitis) involves the scalp and hair; patches similar to the above are formed, only their formation is accompanied by changes in the hair itself. The hair loses its lustre, looks dusty, breaks easily, falls out, and bald spots soon form at the sites of the lesions.

Ringworm of the nails (tinea unguium). Greyish or yellowish spots first appear in the depth of the nails; they grow rapidly and soon involve the entire nail, making it lose its shine, become dull, cloudy, dirty-grey or yellowish. Subsequently the infected nails become deformed, rough and bumpy; they break and crumble easily.

Ringworm is transmitted from infected to healthy children during play, and also from infected domestic pets (cats and dogs). Infection may also be transmitted through various objects the infected person has been handling.

Diagnosis is determined on the basis of the described characteristic lesions of the skin and on microscopic examinations of the scales and hairs in which the fungi mycelium has been discovered.

Prognosis is good if timely and proper treatment is instituted. Ringworm of the nails responds poorly to treatment, and the process may continue for years. The best results are obtained with ringworm of the body.

Treatment. In body ringworm the affected areas are painted with a mixture of tincture of iodine and tincture of gall-nuts (1:1):

Rp. Tincturae jodi
Tincturae gallarum aa 10.0
DS. For external use

This preparation is applied for three to five days; if treatment is begun early the redness in the centre of the lesion disappears and recovery soon follows. When the hair is involved it should be removed by X-rays (this applies only to children over three years old) or by the application of a paste containing 5 to 20 per cent of thallic acetate.

After the removal of both infected and healthy hair the iodine-gall-nut tincture and Wilkinson's ointment (a compound sulfur ointment) are applied to the bare scalp on alternate days; the course of treatment is 14-16 days, and the scalp must be scrubbed and washed every other day. Precautions should be taken in caring for and treating such patients, as the infection may easily involve the nurse's nails. After contact with infected individuals the hands must be washed thoroughly with soap and water, and then in a 5 per cent solution of carbolic acid, or a 1 : 1,000 solution of mercury bichloride; it is still better to wear rubber gloves, particularly for washing the head.

SCABIES

Scabies, or *itch*, is observed in children of all ages. It is caused by infestation with the itch mite *Sarcoptes scabiei* (*Acarus scabiei*). The female mite burrows into the skin, forming "runs" and causing intense itching; scratching causes secondary infections, vesicles and papulae erupting along the runs, most commonly between fingers and toes and on the soles of the feet, as the child rubs one foot against the other to ease the itch. Healthy children get the itch from infested children, or from infested people who care for them, or through clothing and bedding. Unless it is treated, scabies can go on for a long time, spreading over the skin until a large part of the body is involved.

Prophylaxis. Children should be protected from contact with infected persons, they should never sleep on linen used by other people, or wear other children's unlaundered underwear.

Treatment of the infected child alone is not enough. The people he lives with and comes into contact with must be examined for scabies and simultaneously treated, and bedding and clothing must be disinfected to avoid reinfection. The most common preparation used is Wilkinson's ointment mixed with petrolatum jelly (Vaseline) in equal proportions, or sulfur ointment (*Ung. sulfuratum*), rubbed into the involved areas twice a day. After three days the child is given a bath, his underwear and bedclothes are changed,

his mattress and pillow disinfected. If runs are still visible the treatment is repeated. Good results are also obtained by the following method: a solution of hyposulfite (sodium thiosulfate, 50 g to 150 ml of water) is applied to the involved areas, then the child is left unclothed for 15 minutes and a 3 per cent solution of hydrochloric acid is applied to the same areas and left on for 45 minutes. The procedure is repeated from the beginning after the 45 minutes have elapsed. Thus, the entire treatment takes approximately two and a half hours.

DISORDERS OF THE DIGESTIVE ORGANS

Diseases of the mouth and of the gastrointestinal tract make up 35 to 40 per cent of all the diseases of childhood. The mortality rate due to these disorders is 25-35 per cent of the total child mortality rate. Sickness and mortality are mostly observed in early childhood (up to two years).

CATARRHAL STOMATITIS

This form of stomatitis is observed most commonly in infancy. It is characterised by the appearance of red swellings on the mucous membrane of the mouth and particularly of the gums; the latter may even bleed, and excessive saliva secretion is almost always noted. Infants do not take the breast readily, older children complain of pain when they chew and swallow food; in some cases the patients lose their appetite and run a slight temperature.

Catarrhal stomatitis is not a dangerous disease, and it calls for no special treatment. It is sufficient to wash out the mouth with a solution of potassium permanganate (1:5,000) and dust the oral mucosa with powdered sugar or apply sugar syrup to it.

APHTHOUS STOMATITIS

Aphthous stomatitis is a form characterised by the presence of peculiar small vesicles on the hyperemic mucosa of the mouth (the cheeks, lips, tongue and less often the soft palate); the vesicles are usually the size of a lentil seed and they are encircled by a narrow white rim that sometimes has a reddish edge. These vesicles may merge into larger lesions. Stomatitis of this kind is contagious, and it often spreads from one child to another.

The pathogen of aphthous stomatitis is unknown; some authors consider this disease to be analogous with the aphthae of cattle (the foot-and-mouth disease); if so, it is possible that the infection is transmitted through milk.

Clinical findings. Besides local symptoms, in some cases sharply expressed and accompanied by pain and saliva-secretion, more or less severe generalised disorders may be observed, in most cases with high temperatures (up to 40°).

In *diagnosing* aphthous stomatitis attention should be paid to the possibility of confusing it with oral diphtheria, particularly in cases when the aphthae merge into large spots. In these cases diagnosis is facilitated by the finding of separate typical vesicles located at a distance from the merged crops, but sometimes it is necessary to make a bacteriological examination.

The duration of the disease is one to two weeks; complete recovery usually occurs. However, in feeble infants the disease may be complicated by ulcerations, swelling and cracking of the lips, irritation of the skin of the face.

Prophylaxis consists mainly of protecting healthy children from contact with infected ones. The latter should have separate dishes and utensils that are washed thoroughly after each meal. As regards babies, their nipples, toys and other objects they may put into their mouths must be scrupulously clean.

Treatment. The mouth is usually rinsed or sprayed with a solution of potassium permanganate (1:2,000) and a 2 per cent solution of hydrogen peroxide or rivanol.

Rp. Sol. Rivanoli (1:500) 100,0
DS. 1 teaspoonful per glass of water
for rinsing the mouth

In cases that do not respond easily to the above washes and sprays it is recommended to apply, cautiously, a 2 per cent solution of silver nitrate to the aphthae (but not to the entire mucosa). The patient must be given liquid, nonirritating and never hot food.

ULCERATIVE STOMATITIS (TRENCH MOUTH OR VINCENT'S ANGINA)

This form of stomatitis is observed in children of a more advanced age, not before the eruption of their teeth, and particularly in the presence of tooth decay (caries). It begins with the gums swelling, becoming inflamed and painful. The gingiva (the oral

mucosa surrounding the teeth) becomes purulent, necrotic; ulcerative degeneration of the gums follows, and the roots of the involved teeth are disclosed. The teeth often become loose. The infection may then spread to the other side of the mouth. The patient's breath is foul and he secretes saliva abundantly. Microscopic examinations of the pus and ulcerative matter show various spirochetes and spindle-shaped bacilli usually associated with dental caries; possibly, these are the etiological factors of the disease, particularly in cases where the organism was weakened by a previous illness.

Ulcerative stomatitis is more common in cachectic children, or in children recovering from some infection.

Ulcerative stomatitis produces, as does aphthous stomatitis, general systemic symptoms: elevation of temperature, pains, and in some cases a swelling of the lymphatic glands. However, notwithstanding the fact that the disease is often very violent, the process usually ceases after a week or two, and the ulcers heal.

In *differential* diagnosis mercurial stomatitis should be taken into consideration; this form of stomatitis is observed in syphilitic children during treatment with preparations of mercury. Another form of stomatitis is associated with scurvy—scurbutic stomatitis.

Prophylaxis includes mouth hygiene during the child's illness (rinses, frequent drinks). Dental treatment of carious teeth and care of the teeth is also important.

Treatment is the same as for aphthous stomatitis. In severe cases it is recommended to powder the affected places with sulfonamides.

Ulcerative stomatitis is more often observed in undernourished and in cachectic children, and therefore the institution and proper management of a full-value dietary regimen with sufficient vitamins (fruit juices, butter) is essential.

GANGRENOUS STOMATITIS

This form of stomatitis is a gangrenous disease of the mucous membrane of the mouth; it starts in debilitated children during or following certain infectious diseases (measles, dysentery, typhoid, less often after other infections); it is also associated with various types of dystrophy. The lesions of the mucosa may become associated with gangrenous lesions of the jaw bones, the bones

of the nose or hard palate. Certain severe forms of gangrenous stomatitis affect the entire tissues of the cheeks or lips up to the skin.

The disease is usually observed in children over one year of age; it is characterised by the appearance of gangrenous degenerations on the affected areas accompanied by a specific foul odour; the colour of the necrotic mass of decomposing tissues is usually grey or greyish-green; in cases when all the layers of soft tissues and skin are involved the gangrene is almost black.



Fig. 27. Gangrenous stomatitis (noma) in four-year-old boy

At present it is considered that the pathogen of this disease is a group of anaerobic microbes, the most pathogenic of which are *Bac. perfringens* and the anaerobic streptococcus.

This type of lesion may appear spontaneously or it may develop from a preceding ulcerative stomatitis.

Gangrenous lesions of the mucous membrane associated with simultaneous or subsequent lesions of the entire thickness of the soft tissues of the oral cavity, reaching the skin (moist gangrene of the skin) is called *watery cancer* or *noma* (*cancrum oris*) (Fig. 27).

The foul odour that accompanies noma is so strong that it is felt immediately upon entering the room where the patient is. The colour of the gangrenous matter is dark, even black. The disease and degeneration of the tissue follows a very rapid course. In some cases the bone tissue also becomes involved (the jaws, the hard palate, the nasal bones).

Prognosis in gangrenous stomatitis depends on the extent of involvement.

Treatment. In all forms of gangrenous stomatitis the serum commonly used for the treatment of gas gangrene is administered. This serum is prepared against four sources of anaerobic infection: *Bac. perfringens* (*Clostridium perfringens*), *Bac. oedematiens* (*Clostridium novyi*), *Vibrio septicus* (*Clostridium septicum*), and *Vibrio histolyticus* (*Clostridium histolyticum*).

The amount of serum is computed in antitoxin units; the compound serum prepared in our bacteriological institutions contains 5,000 u. All forms of gangrenous stomatitis are treated by 20,000 u on the first day; later the single dose of serum may be lowered to 10,000 or even 5,000 u, depending on the degree and severity of the disease. The serum is injected for three to four, and at times even six consecutive days (the latter for noma). If the lesions are not too marked 40,000 to 50,000 u are usually enough for one course of treatment, a more severe type of lesion requires 50,000 to 80,000 u for the entire course, while 100,000 u and more are administered in the treatment of noma.

In addition to serum injections local therapy is also employed—the oral cavity is washed out and rinsed with solutions of potassium permanganate (1:5,000), rivanol (1:1,000), hydrogen peroxide. Moreover, the lesions are powdered with pulverised sulfo-namides, or aniline dye tinctures are applied (a 2 per cent solution of methylene blue or gentian violet, or a 1 per cent solution of brilliant green). Good results have been obtained by spraying the mouth with solutions of penicillin.

Thorough mouth hygiene is very important (rinsing the mouth, particularly after meals).

In gangrenous stomatitis sufficient amounts of vitamin C (ascorbic acid, dogrose hip extract) and vitamin A (fish-liver oil or vitamin A preparations in oil) should be administered.

The child's food must be of full dietary value (meat, butter, milk).

Prophylaxis consists of scrupulous care of the mouth during any infectious diseases, of providing good general hygienic measures in caring for and feeding children.

THRUSH (SOOR OR MONILIASIS)

Thrush is a common fungus infection of the mouth of infants, particularly of infants weakened during or after some illness. It may affect healthy babies if their mouths are wiped out carelessly,

or if they are given pacifiers that are not kept clean enough. In children of a more advanced age, and in adults as well, thrush may appear during severe diseases like typhoid, dysentery, or other emaciating illnesses.

The fungus that evokes the disease is called *Candida* (or *Monilia*, or *Oidium*) *albicans*; the infant contracts the spores of this fungus from the air or contaminated objects or fingers; penetrating the mucous membrane of the mouth, the spores start growing and form white patches consisting of filaments of mycelium and conidia of this fungus. The mucosa reddens, then small white vesicles appear, the vesicles later merge into white patches looking like milk scum or curds; these patches may involve large portions of the oral mucosa and even spread down into the esophagus and stomach. The whitish scum does not wipe off easily; if it is rubbed off the underlying skin bleeds and looks inflamed, and the white spots again appear in the same place.

Moderate cases of thrush do not evoke any unpleasant sensations in the patient. Infants nurse and swallow normally; however, if the cheeks and tongue and roof of the mouth are all completely involved, particularly if thrush has spread to the throat, sucking and swallowing become painful and infants do not take the breast readily.

Diagnosis is determined on the basis of the nature of the white patches on the oral mucosa, and it may be confirmed microscopically.

Prognosis is good; under proper treatment (in healthy children even without any treatment at all) the symptoms rapidly disappear.

Treatment usually consists of applying to the parts involved a solution of borax in glycerin:

Rp. Boracis 2.0
Aq. destill.
Glycerini aa 10.0
DS. For applying to the inner surface of the mouth
1-2 times a day

A more energetic measure is the application of solutions of certain aniline dyes (0.5-1 per cent aqueous solution of dahlia violet or 1 per cent solution of gentian violet), or of a 0.5 per cent solution of tryptaflavin; it is usually sufficient to apply the above preparations to the mucosa one or two days for thrush to disappear.

Some authors recommend treatment of protracted cases by

powdering the oral mucosa with pulverised sugar (or applying sugar syrup) and cauterising the affected areas with a 1 or 2 per cent solution of silver nitrate. A suspension of nystatin (100,000 u/ml) is also used (10 drops applied to mouth four times daily after ingestion of food or milk).

Prophylaxis of thrush consists mainly of the strict observance of hygienic rules in feeding infants (frequent hand washing, keeping the mother's breast perfectly clean, sterilisation of pacifiers, nipples, and bottles). The infant's mouth should never be swabbed (to avoid scratching the mucosa) without special orders from the doctor.

RETROPHARYNGEAL ABSCESES

This disease is more commonly observed in early childhood (before two years). Usually the abscess is caused by an infection of the pharynx and nasopharynx (influenza, measles, scarlet fever).

The *anatomic* location of such abscesses is the tissue between the pharynx and the spinal column; in some cases it is the lateral wall of the pharynx behind the tonsils that is involved; the abscess usually begins with an inflammation of the lymphatic glands in the tissues; subsequently, the glands soften and form an abscess.

The *clinical* course of the abscess is violent. The principal symptoms of retropharyngeal abscesses are: hoarse breath developing into a wheezy suffocative type of respiration, difficulties in swallowing; if the disease progresses, rigidity of the neck is observed, and the head is slightly thrown back.

Final *diagnosis* is determined by palpation of the back of the throat through the mouth.

In cases of commencing lymphadenitis a hard infiltrate may be felt in the middle of the back of the throat, or slightly to one side. Subsequently, during the formation of the abscess, a fluctuating swelling is felt in this place. Large high abscesses are sometimes visible to the eye. In some cases a swelling is observed on the neck at the posterior margin of the sternocleidomastoid muscle, due to the enlargement of the gland.

In diagnosing retropharyngeal abscesses it must be remembered that there is a possibility of the abscess being a cold edematous abscess that may develop in this site in tuberculosis of the vertebral column. However, while retropharyngeal abscesses run a very violent course with a high temperature (40° and more), tuberculous abscesses usually develop very slowly, with no significant elevations of temperature, or with normal temperatures.

Prognosis depends on the timeliness of the measures taken (lancing the abscess). When left to itself, the disease may terminate lethally owing to suffocation; in spontaneous ruptures of the abscess the pus may be aspirated into the trachea and also be the cause of death, or lead to the development of pneumonia.

Treatment. At the beginning of the disease only observation, rinses, and abundant drinks; penicillin is sometimes effective in resolving the abscess. Fluctuation (softening of the abscess) calls for immediate incision through the mouth by means of an ordinary lancet wrapped almost to its tip in adhesive tape or gauze. The child's head should be bent forward at once to avoid inspiration of pus.

This operation usually evokes rapid improvement, the temperature falls, breathing is restored to normal, and the child recovers. It is only in extremely rare cases that a second operation is necessary, when the opening closes too soon.

BURNS OF THE ESOPHAGUS

Children may sustain burns of the esophagus by swallowing alkalis (caustic soda), or, less frequently, some acids (strong vinegar).

Little children sometimes burn their throats and gullets by swallowing hot milk too fast.

Almost all burns of the esophagus (gullet) are accompanied by burns of the mouth and throat. Strong solutions of caustic soda first evoke a swelling, followed by necrosis and ulceration and subsequent stricture of the esophagus due to scarring. The clinical symptoms of this kind of burn are vomiting, difficulty in swallowing hard food; stricture of the esophagus is clearly defined by probing.

Treatment. A child who has swallowed some caustic alkali (lye, ammonia, washing soda, potash, caustic lime, quicklime, caustic soda) must immediately be given a mild dilute acid to drink (acetate [vinegar], citrate [lemon], or hydrochloric acid) to neutralise the alkali; if the poison was acetate or another acid he is given a solution of soda or magnesia. The child must be taken to the nearest hospital or polyclinic as fast as possible. If the burn was caused by an alkali gastric lavage is indicated, using 2.5-3 l of warm water to which 100 ml of 10 per cent hydrochloric acid has been added. For burns caused by acids gastric lavage is performed with a 2 per cent solution of soda. If the child is very restless and suffers sharp pains, a 0.5-1 ml injection of 1 per

cent solution of pantopon is given prior to lavage. The involvement of large areas of the mucosa of the mouth and throat in the burn are a contraindication to gastric lavage. Following gastric lavage the child must be kept absolutely at rest in bed (in the hospital). In severe cases blood transfusions are indicated (50-100 ml depending on age), and the intravenous injection of 20-40 ml of a 20 per cent solution of glucose and 50 ml of physiological salt solution. Alkali poisoning is treated with a 1 per cent solution of dilute hydrochloric acid (1 per cent Sol. acidi muriatici diluti); one teaspoonful of this solution and a half teaspoonful of fish-liver oil are given orally every two hours. For prophylactic purposes it is recommended to spray the oral mucosa every two hours with 10 to 15 drops of a solution of penicillin (25,000 u to 1 ml of physiological salt solution). Weakening of cardiac activity calls for the injection of caffeine or camphor subcutaneously. During the first two days after the burn the child is given only liquid food: kefir, kissel (fruit juice starch pudding), broth, liquid wheat cereal; on the following days a semiliquid diet is prescribed (no bread); on the fifth to seventh day the child may be given ordinary meals suited to his age. If only liquid food is given for a prolonged period this facilitates constriction of the esophagus, while rougher food prevents, to a certain extent, a rapid formation of strictures.

After the temperature has gone down, on the third to seventh day of the illness, cautious bouginage (dilatation) of the esophagus is commenced; at first a very slender bougie is introduced, and a thicker one is inserted every subsequent time. Early dilatation of the esophagus prevents the formation of rough scar tissue that obstructs the passage of food. Dilatation is not performed if the patient is running a high temperature or if his general state is bad.

The above measures are mainly applied for burns of the esophagus caused by alkali, occurrences more common than burns incurred by swallowing acids. In the latter cases (mostly strong vinegar) it is usually enough to lavage the stomach on the day of the accident, and to give the child calcined magnesia (20.0-200.0), milk of magnesia, egg white, mucoid cereal water and the above-described liquid diet with a large quantity of fats (fish-liver oil, cream, milk). It is only in severe cases of poisoning with vinegar essence, strong hydrochloric or sulfuric acid that significant lesions are formed on the mucous membranes of the mouth and esophagus, to be followed by a subsequent development of rough scar tissue that requires the same treatment as in poisoning with caustic alkalis.

ACUTE GASTRITIS*

Acute gastritis is a disease in which the prevalent symptoms, besides a general state of malaise and sometimes headache (intoxication) are gastric symptoms (vomiting, pains). The cause of this disorder may be overfeeding or bad food (unripe or spoiled fruit and other products).

However, acute gastric symptoms may also appear when the food is normal; the condition is then presumably due to some infection that penetrates into the stomach with the food, although in each given case it is difficult to differentiate between violation of dietary rules and infection. The symptoms of gastritis (vomiting, pain in the stomach and general indisposition) are usually present for a short while, and recovery soon takes place. If there are persistent pains in the substernal region, particularly at night, it is essential to consider the possibility of gastric or duodenal ulcers and to make an X-ray examination.

Gastric symptoms may also be of a parenteral nature associated with any infection (measles, influenza); in these cases the gastritis passes away upon recovery from the infection.

Frequently diarrhea follows the gastritic symptoms, and the disorder then develops into gastroenteritis.

Treatment of gastritis. Hunger diet on the first day (only tea and water are given), and warmth to the abdomen (hot-water bottles or warming compresses). A light diet is prescribed on the second day: thin cereal, kissel, tea with toast; other foods are added on subsequent days and the patient is allowed to have his usual food depending on the course of the disorder.

Anodynes (opium preparations) are rarely needed for children. Usually the pains disappear with the application of warmth and the modification of diet.

ACUTE GASTROENTERITIS

Acute gastroenteritis is an acute disturbance of digestion accompanied by general manifestations of intoxication, gastrointestinal symptoms being prevalent (diarrhea, vomiting, pains).

* According to the approved classification of acute digestive disorders in childhood gastritis and gastroenteritis are diseases of children older than two years. In infants and babies younger than two years these diseases are looked upon as ordinary dyspepsia and toxic dyspepsia (see p. 147).

The cause of the disorder is the same as for acute gastritis. In the majority of cases it is an infection, frequently with paratyphoid bacilli.

There are no noticeable anatomic changes in the stomach and intestines except hyperemia and swelling of the mucosa.

The course of the disease may be somewhat more protracted than in acute gastritis, but it is not a very long one; recovery is rapid following the administration of corresponding measure.

Treatment is similar to treatment of acute gastritis: hunger on the first day, a light diet on the following days (see "Acute Gastritis"), then a restricted diet for several days (no rough foods such as brown bread and large amounts of vegetables and fruits).

Children should not be kept too long on a restricted diet. As soon as the gastroenteritic symptoms disappear the child must be given the usual food suitable to his age. If there are pains in the stomach warmth is applied (hot-water bags, warming compresses). As in gastritis, anodynes are usually not given; significant meteorism (gaseous distention of the abdomen or intestine) may cause pains in the stomach; in this case pulverised animal charcoal is quite effective: Carbo animalis 1.0 g several times a day.

It is usually not recommended to administer the so-called disinfecting drugs (salol, benzonaphthol), as their effects are dubious. Astringent agents like bismuth and tannin are of a similarly doubtful nature. If there is any suspicion of paratyphoid a bacteriological examination of the feces and the Widal test should be performed, and the child isolated.

COLITIS

Colitis is the term applied to diarrheas accompanied by a characteristic excretion of mucus. Mucous feces are often observed in association with numerous intestinal infections and are no independent intestinal disorder, but only one of the symptoms of a disturbance. It is only in dysentery that this symptom prevails over all others from the very onset of the disease, and it is the decisive factor in diagnosing the disease; moreover, the stool may contain streaks of blood. In such cases colitis (lesion of the large intestine) is a primary process evoked by the action of the dysenteric toxin on the mucosa of the large intestine.

The question of the possibility of primary colitis being of a nondysenterial source is still a moot one. Many authors reject this possibility and consider that in the overwhelming majority of cases all primary colitis of an epidemic nature are nothing else

but *dysentery*. Bacteriological examinations show that in most cases of this type of colitis the dysenteric microbes are discovered (Shiga-Kruse, Flexner, Sonne, and other bacilli).

From a practical standpoint this view is the most correct one, both as regards isolation and treatment, and sanitary propaganda for combating this type of diarrhea in children. Observations have shown that frequently children's summer diarrhea accompanied by the passage of mucus is followed by outbursts of dysentery among the adult population, and, contrarywise, during attacks of dysentery among adults and older children mild forms of this disease are not unusual among babies. If dysenteric colitis in very young children may be called primary colitis, then all other intestinal lesions characterised by the passage of mucus or of mucus and blood in the stool during certain illnesses in children, particularly during measles, whooping cough and influenza, may be called *secondary colitis*. The stools of young children contain mucus, or sometimes mucus and blood during such infectious diseases as typhoid and paratyphoid, being one of the principal symptoms of the disease. This type of diarrhea is not colitis, and, just as dysenteric colitis is simply called dysentery, so are such intestinal lesions in children looked upon as manifestations of typhoid or paratyphoid.

Anatomic findings in secondary colitis show the swelling chiefly of the mucosa of the large intestine accompanied by a secretion of large amounts of mucus, and in some cases by a significant involvement of the follicular apparatus of the intestines; fibrinous-necrotic changes in the mucous membrane of the intestines with a subsequent formation of ulcers is much rarer.

Bacteriological examinations in secondary colitis reveal the most diverse flora, often differing little from that of children who pass no mucus with their stools.

Thorough bacteriological examinations made during colitis attending or following measles usually show true dysenteric bacilli. Evidently, the primary infection (measles) weakens the resistance (immunity) of the organism and prepares the ground for a secondary infection (dysentery).

Clinical findings. The clinical aspect of the disease in secondary colitis is variant, ranging from very mild cases with rapid recovery to severe prolonged cases terminating fatally. This depends to a large extent on the severity and duration of the basic disease. But in some protracted cases of colitis followed by subsequent non-protein edemas the course and severity of the disease are due to bad therapy.

Treatment. At present the treatment of colitis differs little from that of dysentery: first a short hunger period (from several hours to a day or two) followed by a nutritive, easily assimilated, diet. Children are given sieved cereals, puréed vegetables, ground cooked (but not fried nor roasted) meat, the only fats during the first days are cream (with roasted grain beverages or tea), white bread and soaked toast (in moderation), also dairy products such as curds (cottage cheese) and kefir; sieved or grated cooked and raw fruits, kissels (thin starch puddings); vitamins are provided with fruit juices (lemon, carrots). Sulfonamides, synthomycin and a number of other drugs are administered.

Pains in the abdomen call for the application of warmth (hot-water bags, compresses, hot baths). If the necessity arises in severe cases the usual cardiovascular agents are resorted to (caffeine, camphor).

Prophylaxis of secondary colitis is general care of the basic infection and expedient therapy.

Prognosis depends on the severity of the primary infection and on the conduction of all the necessary measures in caring for and feeding the child; prognosis is good if proper measures are strictly adhered to.

PERITONITIS

Acute inflammations of the peritoneum affect children of any age.

Purulent peritonitis. Etiology and pathogenesis. Acute purulent peritonitis* is a result of the penetration of pathogenic microbes into the peritoneum by different routes.

As in adults, peritonitis may be the outcome of injuries, of accidental abdominal wounds, but in children it is mostly caused by infection carried from neighbouring parts (appendicitis, gonorrheal infection of the sex organs in girls, etc.). Another source of infection are perforating intestinal ulcers that may develop in certain diseases (typhoid, dysentery, tuberculosis).

However, particularly in young children, the infection may often be carried to the peritoneum by the lymph or blood stream during various septic diseases, thus constituting a partial symptom of septic pyemia. This type of peritonitis may be observed as a complication during or following many infectious diseases (pneumonia, erysipelas, diphtheria, scarlet fever).

* See chapters on tuberculosis and rheumatism for serous peritonitis.

Pathologic anatomy. Postmortem examinations show a characteristic red inflammation of the peritoneum which is permeated with a dense vascular network and coated with a cloudy exudate. In purulent peritonitis the exudate consists of thick or thinly dispersed pus, sometimes (in perforating peritonitis) the pus looks dirty and its odour is fetid. The intestine is usually extended, often the loops adhere to each other. In benign courses of peritonitis the exudate may become encapsulated in a pouch, and the pus may then break out through the navel, or into the intestine, vagina, or bladder. Upon recovery connective tissue is formed in place of the pus; this tissue is at first soft and vascular, subsequently it solidifies and hardens.

Clinical findings in peritonitis in children are of the same general aspect as in adults: vomiting, constipation, abdominal tension, distension and tenderness, wasted face (*facies hippocratica*), sunken eyes, rapid pulse. However, in children, particularly very young ones, the abdomen may reveal no or very little tenderness. In certain cases purulent peritonitis in children may terminate by the pus breaking out through the navel, anus or vagina. No sharp abdominal pain may have been observed prior to this in the child.

At the onset of the disease a significant elevation of temperature is usually observed (40° and higher), the temperature keeping to one level or becoming slightly lower at times. The course of the disease is a very violent one, and very young children often succumb within 36 to 48 hours. At the same time local abdominal symptoms may be absent, and in these cases the only symptoms are those of generalised intoxication.

In less violent courses of peritonitis the local abdominal symptoms are observed to develop gradually: the stomach is at first somewhat depressed, and in some cases it is very hard owing to a general contracture of the abdominal muscles; later abdominal distension appears and dulled sounds are evoked on percussion of the sloping areas of the abdomen owing to a large accumulation of exudate.

Older children complain of sharp pains that are intensified by coughing and moving. If the child does not succumb in the first days to generalised (or diffused) peritonitis the pus begins accumulating in definite areas, mainly in the vicinity of the navel and in the lower part of the abdomen; the pains become weaker, and when the pus has either found a natural outlet or been removed surgically the child recovers.

All forms of purulent peritonitis follow different courses, depending on the causative agent.

Pneumococcal peritonitis. Its symptoms are the usual symptoms of purulent peritonitis, but its course is in many cases more benign than that of streptococcal peritonitis. Encapsulation of the pus is

often observed in pneumococcal peritonitis; a subsequent natural or artificial (surgery) elimination of the pus leads to complete recovery in most cases.

An independent form of pneumococcal peritonitis is sometimes observed; its initial symptoms often simulate dysentery, appendicitis, volvulus (intussusception or invagination of the intestine).

Peritonitis owing to perforation of the stomach or intestine is rarely observed among children. The chief symptoms of this form of peritonitis are a sudden onset of sharp pains in the abdomen, often accompanied by collapse, a running pulse, rapid elevation of the temperature to 40° and more, a characteristic yellow-green vomitus (sometimes brownish-black) with a fecal odour. The disease runs a rapid course and terminates fatally.

Streptococcal purulent peritonitis is observed as a complication of scarlet fever and other diseases; its course is a very violent one, accompanied by the usual symptoms of peritonitis and the formation of a purulent exudate in the peritoneum. Its termination is fatal.

Gonococcal peritonitis in girls with gonorrheal affections of the sex organs is encountered very seldom; it runs a comparatively benign course and is accompanied by general symptoms of peritonitis; its duration is short (two to eight days) and recovery usually occurs.

Peritonitis of the newborn is often the result of diseases of the umbilicus, but in some cases it is a symptom of general sepsis, whatever its origin. Peritonitis is diagnosed in the newborn mainly on the basis of other septic displays, when local symptoms in the abdominal area are insufficiently expressed; often on postmortem examinations of newborn infants who have succumbed to septicemia pus is found in the peritoneal cavity, while there were no symptoms of peritonitis during life. One of the symptoms of peritonitis in the newborn is edema of the skin over the abdomen, sometimes jaundice, meteorism, and vomiting attendant on a generalised pattern of sepsis.

Prognosis depends on the etiology of the disorder. Recovery is observed in certain cases of pneumococcal peritonitis.

Treatment. Purulent peritonitis of any etiology whatsoever requires absolute repose at the beginning of the disease, an icebag to the abdomen, the infusion of normal saline solution, penicillin intramuscularly, sulfa drugs orally. If peritonitis is suspected a surgeon should be consulted immediately. Surgery is not indicated for all forms of peritonitis, it is only feasible in cases when the pus has encapsulated in the peritoneum, usually only in pneumococcal peritonitis. Surgery for all other general forms of peritonitis is usually unsuccessful. Surgery is indicated for intestinal perforation. There is no need for surgery in gonococcal peritonitis.

HELMINTHIASES (WORM DISEASES)

Man is host to as many as 135 species of parasitic worms—helminths. They infest various sections of the intestinal tract, sometimes only the rectum; certain helminths (trichinae) infest muscu-

lar tissues, the liver, the lungs, and brain (echinococcus.) The parasitic worms most widespread among children are described on the following pages (Fig. 28).

Pathogenesis. Through soil, sand and water the egg-containing feces of worm hosts contaminate vegetables and fruits; the eggs (ova) thus infest children. The disease may also be contracted through sleeping in the same bed with a worm host.

Some helminths (tapeworms) have, besides their human host, an intermediate animal host as well; these worms may be ingested with undercooked meat or fish.

Clinical findings. Parasitic worms are the cause of diverse disorders and lesions in the child's body. The toxic substances secreted by helminths, and the itching they cause may lead to nervous disorders; by penetrating into the intestinal mucosa they open the way for various pathogenic microbes. Symptoms of worm diseases vary considerably. Sometimes the patient loses his appetite completely, in others he cannot eat his fill*; nausea, vomiting, pains in the epigastric region may be present, and in some cases diarrhea; at times the child sleeps restlessly and his nervous excitability increases. Worm infestations may lead to anemia and malnutrition; eosinophilia is often noted (5 to 10 per cent of eosinophils in the blood count).

However, helminthiasis frequently follow no definite clinical pattern; most of the above-mentioned symptoms may be absent and the child appears healthy. Diagnosis is then only possible if worms are passed with the stool, or if helminth ova are detected in the feces by microscopic analysis.

Prognosis. Notwithstanding the apparently very bad effect of parasitic worms on the organism, prognosis is quite good if timely measures are taken for ridding the child of worms. It is only in very neglected and far-gone cases that acute anemia and malnutrition develop.

Prophylaxis. The general prevention of the spread of worm diseases among children and the population at large consists of measures for preventing the contamination of toilets, various objects and food with helminth ova. Personal prophylaxis is also important. The following precautions are recommended:

Wells or other types of water reservoirs from which drinking water is obtained should be located as far as possible from toilets and covered with lids to protect the water against various impurities.

* If this symptom is present the urine should be tested for sugar, as diabetes is usually accompanied by a constant feeling of hunger.

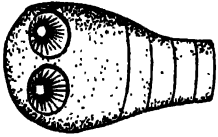
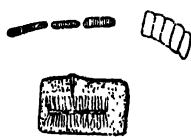

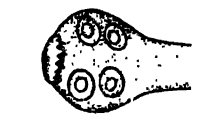





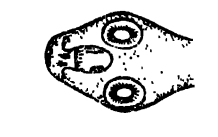


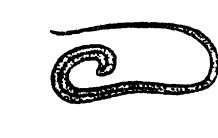






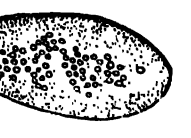




	Adult specimen(head)	Segments	Eggs
<i>Ascaris</i>			
<i>Taenia saginata</i>			
<i>Taenia solium</i>			
<i>Bothriocephal lat</i>			
<i>Taenia nano</i>			
<i>Trichocephal dis</i>			
<i>Oxyuris vermic</i>			
<i>Taenia echinoc</i>			

Fig. 28. Parasitic worms most common in man, and their ova device for oxygen therapy in ascariasis.

Worms ejected from the body (either spontaneously or by means of vermifuges) must not be disposed of in toilets or drain pits; they should be burned or scalded with boiling water.

Children must be instructed in rules of personal hygiene, be taught to use toilet paper, to wash their hands after visiting the toilet and before meals, not to bite their nails or suck their fingers.

Flies must be destroyed and food protected against them.

Children should not be allowed to fondle domestic pets (cats, dogs).

Children should not be given unboiled water to drink, nor have unwashed vegetables and fruits; their meat and fish must always be cooked thoroughly.

ASCARIASIS (ROUNDWORMS)

The species of roundworm or ascarid that causes ascariasis is called *Ascaris lumbricoides*. The male is 15 to 20 cm long, the female 25 to 40 cm. A child may be host to quite a large number of these earthworm-like parasites (from 10 to 100). The ascarid eggs (or ova) are excreted in feces, then the highly infective larvae develop in them in moist soil, within 12 to 40 days (at temperatures ranging from 7 to 40°C). Children become infected by ingesting the larvae-containing eggs from soil-contaminated fingers, with unwashed vegetables, berries, or fruits. In the intestine the larvae emerge from the ingested eggs and penetrate through the mucous membrane of the intestine into the blood stream; with the blood they are carried to the portal vein, into the right atrium, to the lungs, through the alveoli to the bronchi, trachea, larynx, pharynx, then are swallowed and thus they again appear first in the stomach and then in the intestine where they finally develop into mature worms. The entire cycle of development takes approximately 75 days.

The following symptoms of ascariasis are observed in children

Gastrointestinal: an aversion to certain foods (most commonly fats), nausea, vomiting (usually in the morning), at times dizziness and faintness; unpleasant sensations in the substernal region, salivation (particularly at night), abdominal pains, commonly in the substernal and navel areas; constipation or diarrhea, abdominal distension. In rare cases ascarids can cause appendicitis, intestinal obstruction or perforation, obstruction of the bile ducts.

Nervous system: irritability, headaches, absent-mindedness, twitching, hysterical or epileptoid attacks. In very rare cases asca-

riasis may cause intoxication of the central nervous system, manifested by meningeal or encephalitic symptoms, producing the clinical aspect of tuberculous meningitis or encephalitis.

Respiratory tract: migrating via the blood stream to the lungs, the larvae may cause bronchitis, or the formation of pneumonic sites in the lungs; at this stage the blood may show an increased eosinophil count (eosinophil infiltration in the lungs).

Diagnosis is established by the demonstration of the ova in the feces.

Treatment. The most effective and widespread method used in the Soviet Union is *santonin* and *sancaphen* therapy. Santonin is administered for two consecutive days in tablets or powders (three times a day); one of the following procedures is employed.

First method: (recommended for older children and for adults). On the first day a light diet and a saline purge before going to bed; on the second and third days three santonin powders on a fasting stomach over one-hour intervals; an hour after the last powder has been taken a laxative is given, and one hour after this a light breakfast.

Second method: (recommended for children younger than four years, and for weak children of a more advanced age). Light diet and saline cathartic at bedtime on the first day; on the second and third days santonin one to one and a half hours before meals and a cathartic at bedtime. The santonin dose for children is 0.005 g per year of life (0.025 g for a five-year old). The bowels are purged of the ascarids by one of the following cathartics: Glauber's salt (sodium sulfate), Epsom salt (magnesium sulfate), or senna tea.

Age (in years)	Glauber's salt (in g)	Epsom salt (in g)	Senna tea (in ml)
2-3	—	—	10
4-5	10	8	15
6-7	12	10	20
8-9	15	12	30
10-12	20	15	35
13-16	25	20	45
17 and older	30	25	60

Children may also be given *compound senna powder* (compound licorice powder) for two consecutive days, once a day, directly after the last santonin dosage. Its doses are: for children from two to three years old half a teaspoonful; from four to six years one level teaspoonful; from eight to sixteen years one and a half to

three teaspoonfuls, and at seventeen years and older four teaspoonfuls.

Diet. A nutritious, light diet is prescribed for one day prior to, during, and one day after treatment; fats are restricted and the food must be easily digested and very soft: soups, semiliquid cereals, sieved meat and vegetables, milk, sour milk (acidified milk); sugar is permitted.

Contraindications for santonin and sancaphen are nephroso-nephritis, acute gastrointestinal and febrile illnesses (not related to ascariasis), pyelitis.

Wormseed treatment. Wormseed (*santonica* or *Flores Cinae*) is administered in a manner similar to the santonin procedure. As every gram of wormseed contains approximately 0.02 g of pure santonin doses for children are: from one to three years 1 g; from four to six years 1.5 g; from seven to nine years 2 g; from ten to fourteen years 3 g; at fifteen years 4 g, and anybody older than sixteen years is given 5 g. The wormseed is chopped and bruised and given in honey, jam, or sugar.

Sancaphen treatment. Commercial sancaphen tablets are very convenient for use in mass and individual deworming. Each tablet contains 0.016 g of santonin, 0.016 g of phenolphthalein and 0.0065 g of calomel.

Sancaphen is given for two consecutive days. The total dose for children is one tablet for each year of their life (for instance, an eight-year old is given a total dose of 8 tablets, or 4 tablets a day); the adult dose is 18 to 20 tablets in two days.

It is recommended to administer sancaphen in the following manner: on the evening preceding treatment a laxative is taken (phenolphthalein, rhubarb tablets or powder, isaphenin), or a purging enema is given; on the following morning before breakfast or an hour after a light breakfast two doses of sancaphen are administered over an interval of one-half to one hour. Breakfast may be had two hours after the tablets. In the evening a laxative or saline purge is given. The same procedure is repeated next day. When sancaphen is used for group deworming in ordinary, trade and vocational schools the laxatives may be given during the last lesson in class, instead of at home in the evening.

During sancaphen treatment spicy, acid, or very fat food is not permitted.

A repeated course of santonin or sancaphen may be given, if required, three to four weeks later.

Santonin, when given to a highly sensitive child, or given in excess may evoke symptoms of intoxication: vomiting, disturbance

of vision (everything looks yellow); in general the urine may become yellow when santonin is ingested.

Piperazine treatment. Piperazine is a very effective agent for treating ascariasis and enterobiasis. It is prescribed for oral administration as an aqueous solution or in sugar syrup.

For ascariasis piperazine is given on two consecutive days, 2-3 times a day. Its dosage is:

Age (in years)	Single dose (in g)	Number of doses per day
1	0.2	2
2-3	0.2	3
4-6	0.5	2
7-9	0.5	3
10-14	1.0	2
15 and older	1.0	3

The preparation is taken 1/2 to 1 hour after meals. After the full number of doses has been administered a laxative is prescribed (phenolphthalein, rhubarb, salts). No special diet is required during treatment. Piperazine is well tolerated, no side effects have been noted when the above therapeutic doses were adhered to.

Oxygen treatment. Currently the evacuation of roundworms by means of oxygen has become quite popular; it is particularly recommended in cases where other vermifuges are contraindicated.

The oxygen is introduced into the stomach through a thin tube; it is delivered from its container through two glass jars connected by a rubber tube; one of the jars is full of water, then the water is pumped into the other, into the first again, etc., the jars serving alternately as meters and pumps.

The child lies on his back, and a thin tube is entered through mouth or nose into the stomach. The oxygen is pumped through the tube in small portions (100-200 ml over 1-2 minute intervals). The entire procedure should take no less than 15 minutes; its acceleration might evoke nausea and pain in the substernal region. For every year of the child's life 100 ml of oxygen is given.

This treatment may be administered both in hospitals and in outpatient clinics. The method is very simple and calls for no complex equipment.

ENTEROBIASIS

Pinworms (*Enterobius vermicularis*, formerly called *Oxyuris vermicularis*) are no longer than 10 mm; they habitate the small and large intestines; the mature females descend into the rectum to lay their eggs in the creases of the anus.

The females usually live no longer than one month; thus a constant reinfection occurs, as pinworm eggs are abundant on the surface of the perineum, around the anus, and also on sheets and underwear. If the child does not wear drawers at night the itching caused by the worms makes him scratch the perianal region; thus the eggs contaminate the fingers, and from there are conveyed to the mouth. For laboratory examination the surface of the skin around the anus is scraped with a blunt instrument, and the scrapings are examined.

Enterobiasis most commonly evokes the following signs and symptoms in children: 1) itching in the area of the anus and genitals; 2) redness and scratches around the anus and genitals that may become sites of a secondary infection (vulvovaginitis, abscesses, eczema; 3) gastrointestinal disorders; loss of appetite, nausea, frequent bowel movements, abdominal pains; pinworms may cause inflammation of the cecum and its appendix (appendicitis); 4) nervous conditions: sleeplessness, night incontinence; often absent-mindedness or lassitude; persistent enterobiasis may lead to severe neurasthenia, and also to masturbation.

Enterobiasis is *diagnosed* on the basis of the finding of the ova; the eggs are to be found only in the mucus scraped from the perianal folds or the lower part of the rectum as the females do not lay their eggs in the intestine. Sometimes the pinworms are seen with the naked eye in the stools; the female worms can also be seen between the buttocks upon parting them slightly; they usually come out to lay their eggs an hour or an hour and a half after the child has gone to sleep.

Treatment. The persistency of enterobiasis is due to constant self-reinfection. Therefore, its treatment can be successful only by observing the following conditions: 1) a systematic course of treatment; 2) simultaneous institution of a number of prophylactic measures; 3) simultaneous treatment of all pinworm-infested members of the family. Enterobiasis is treated with sulfur preparations: precipitated sulfur and sublimed sulfur (*Sulfur praecipitatum* and *Sulfur depuratum*); these preparations are given in equal proportion with licorice powder in a tablespoonful of cereal or vegetable purée. Single doses of sulfur for children are 0.05 g per year

of life (at 6 years 0.3 g) three times a day for five days, then a four-day interval, and again five days of the same treatment. These cycles of five days of treatment and four-day intervals are repeated two to four times; during the intervals an enema is given nightly (one to three glasses of water depending on the child's age); teenagers and adults are given 4-5 glasses of water in the enema.

Rp. Sulfuris praecipitati (seu depurati) 0.5 D. t. d. N. 15
S. 1 powder 3 times a day before meals
(for 5 consecutive days for a child of 10 years)

It is recommended to add half a teaspoonful of baking soda (sodium bicarbonate) per glass of water to the enema. It was formerly recommended to put garlic infusions, salt, tannin, or vinegar in the enemas. At present these substances are not advised as they might irritate the intestinal mucosa and cause straining (tenesmus).

Sometimes sulfur evokes abdominal pains or diarrhea, but these symptoms disappear very soon after the sulfur ingestion has ended.

No special diet is required during sulfur treatment. It is advisable to apply white, yellow or grey mercury ointment to the anus at bedtime, in cases of extreme itching anesthesin ointment is used.

Rp. Anaesthesini 1.0
Vaselini 25.0
DS. For external use

If a child is host to both roundworms and pinworms, the roundworms should be dealt with first (santonin or sancaphen according to the procedures described above).

It is recommended to treat persistent enterobiasis with *Dryopteris Filix-mas* (the European aspidium or male fern) in small doses, similar to the treatment of hymenolepiasis, only without the third cycle (see p. 223). Upon the completion of two cycles the sulfur treatment is administered. Another effective enterobiasis expellant is piperazine. This preparation is given in doses as for ascariasis, in cycles of 3-5 days of treatment and 7-day intervals, a total of two or three cycles.

Measures of individual prophylaxis during treatment: 1) the perineal area is washed morning and evening with soap and warm water, petrolatum jelly is applied abundantly to the perianal region at bedtime; 2) the child, after he has been washed, is dressed in tight drawers or shorts so that he cannot touch the anus; in the morning the shorts are boiled, and clean ones put on for the day; 3) the child should not be allowed to share a bed with other peo-

ple, to avoid mutual pinworm infection; 4) on the first and last days of every course of treatment a) the child is bathed (with soap); b) underwear and bed linen are changed, the soiled linen is properly boiled; c) the blanket and clothing that cannot be boiled or washed are pressed with a hot iron; d) the shoes are cleaned; e) the room is cleaned thoroughly, the floor and furniture wiped with clean moist rags that are boiled afterwards.

TRICHURIASIS (TRICHOCEPHALIASIS)

Whipworms (*Trichuris trichiura* or *Trichocephalus trichiuris*, *Trichocephalus dispar*) frequently infest children's intestines, though less often than roundworms or pinworms. Whipworms are round worms, up to 5.5 cm long; they habitate the lower intestine, chiefly the cecum, boring into the mucosa with their hair-thin front ends. Their eggs are passed with the feces and become fertile outside the human body. Under favourable conditions they may mature within three weeks. Infestation occurs by ingesting fertile whipworm eggs, chiefly with unwashed vegetables, berries, fruits. Infestation is also possible through other foods to which the eggs might have been transferred by flies, dust, or in some other way.

It takes approximately 30 days from the moment the fertile eggs (with living larvae in them) are ingested until the mature female worms lay new eggs. Whipworms usually evoke no particular symptoms, and are only discovered upon examination of feces; however, it does happen that trichuriasis is manifested by a number of dyspeptic symptoms (nausea, heartburn, flatulence), colicky abdominal pains, nervous symptoms (headaches, dizziness, in some cases epileptoid attacks), anemia.

Diagnosis of trichuriasis is determined by the demonstration of the ova in feces.

Treatment. Whipworm infestation is treated with osarsol (also known as acetarsone, acetarsol, stovarsol); this substance is a strong poison and its prescription should therefore be very guarded (healthy state of liver and kidneys); the patient must be hospitalised for the course, and preliminary urinalysis is imperative. Osarsol contraindications are various intestinal disorders, acute skin conditions, hemorrhagic diathesis, acute febrile conditions, diabetes, menstrual periods in girls. The conditions of the kidneys should be watched closely during treatment, and also proper bowel functioning. At the appearance of side effects (fever, headaches, nau-

sea, albumin in the urine and urobilin) osarsol administration is discontinued. It may be resumed when the patient is in a satisfactory state again, but not before five to seven days. If the complications are pronounced, as well as when they recur upon resumption of osarsol administration the treatment must be stopped.

Osarsol is sold in 0.25 g tablets.

In prescribing for children the total osarsol dosage (for a five-day course) is computed on the basis of 0.25 g per year of life. The daily dose is divided into 3-4 smaller doses; for this the tablets must be divided into quarters, thirds, or halves.

Osarsol is very rarely prescribed for children younger than six years old.

Osarsol Dosage

Age (in years)	Single dose (in g)	Total dose (in g)	(5-day course) in tablets
6	0.08	1.5	6
7	0.09	1.75	7
8	0.11	2.0	8
9	0.125	2.25	9
10	0.14	2.5	10
11	0.15	2.75	11
12	0.16	3.0	12
13	0.18	3.25	13
14	0.19	3.5	14
15	0.20	3.75	15
16 and older	0.25	4.0-5.0	16-20

The osarsol tablets are crushed to a powder and given with food for five days; on the morning of the sixth a mild saline cathartic or ordinary purgative enema is given. Osarsol therapy should be administered with great precautions (symptoms of intoxication are fever, roseolous rash, diarrhea, sometimes with blood in the stool, in certain cases brain symptoms). Upon the appearance of such signs osarsol treatment should be stopped and the intestinal tract purged with saline cathartics (Epsom or Glauber's salt, senna) and enemas; a 10 per cent solution of sodium thiosulfate (hyposulfite or Natrii hyposulfurosi) is given every 2-3 hours in doses varying with age (a teaspoonful to a dessertspoonful).

Osarsol therapy may be repeated no sooner than in one month; however, even after this it is often observed that the whipworm eggs do not disappear from the feces.

In cases of repeated intolerance symptoms osarsol treatment should be stopped for a prolonged period.

Trichuriasis deworming results are determined by examinations of the feces made no earlier than 15 days after completing the treatment. If no eggs are discovered in the first analysis it is repeated two or three times over intervals of ten to fifteen days.

TAENIASIS

Taeniasis is a condition caused by infestation with the beef tapeworm *Taenia saginata* (or *Taeniarhynchus saginatus*) or the pork tapeworm *Taenia solium*; these worms exist in the small intestine of man. The beef tapeworm is 4 to 10 metres long, the pork tapeworm 1.5 to 2 m.

The larvae (in the cysticercus stage) of the beef tapeworm infest the muscles of cattle, the pork tapeworm cysticerci live in the organs and muscles of swine, but they are also capable of existing in human organs and tissues (mostly in the brain, eye and subcutaneous tissue). The mature segments (gravid proglottids) are detached from the body of the tapeworm and passed with the feces; the proglottids of the beef tapeworm can also emerge from the rectum independently.

Cattle and swine ingest the eggs or the proglottids of tapeworms with grass or water, and so become infested with the larvae that emerge from the ova in their intestines. These larvae are carried to all parts of the animal's body with the blood, and there they become encysted (turning into cysticerci); they can exist in this manner for several years in the tissues of their intermediate hosts, but to become mature tapeworms they must be ingested by their final host, man. Man usually becomes infested by consuming raw or poorly cooked cystisercus beef or pork; the cysticerci mature into tapeworms in the human intestine two to three months after ingestion. However, if man ingests the eggs of the pork tapeworm (with contaminated water or vegetables) these eggs may turn into cysticerci in his intestine, and the cysticerci (as in the swine) may settle in any of his organs or tissues, causing the condition called cysticercosis; this condition may likewise develop by an internal route if the ova penetrate from the intestine into the stomach of a tapeworm host during attacks of vomiting or nausea.

The clinical aspect of taeniasis is characterised by the gastrointestinal and C.N.S. symptoms listed above in the description of other intestinal worm infestations.

DIPHYLLOBOOTHRIASIS (FISH TAPEWORM DISEASE)

Diphyllobothrium latum, the fish tapeworm, is a large, wide tapeworm 3 to 20 m long. Its plerocercoids (a larval stage) exist in the muscles, fat, liver and kidneys of some species of freshwater fish (pickerel, perch, white-fish, ruff, burbot). Man becomes infested by consuming raw or half-cooked plerocercoid fish. The symptoms of fish tapeworm infestation in children are few; however, general weakness, loss of weight, and severe anemia (even of the pernicious type) may develop.

HYMENOLEPIASIS

Hymenolepis nana (formerly called *Taenia nana*) is the smallest of the dwarf (nana) tapeworms. Its length is only 1.5 to 2 cm. It infests the lower intestine of man, and always in large numbers. The eggs of this tapeworm mature in the intestine and are passed with the feces; they require no time in external environment for maturation, as do the eggs of roundworms (ascarids) and whipworms (trichuris). Slovenly habits are the cause of the eggs contaminating the hands of a host of this worm and thus being spread to various objects and foods. Infestation occurs by ingestion of the ova; in the intestine the larvae emerge from the eggs, and then invade the intestinal villi. The larvae develop into cysticeroids (a second larval stage); the cysticeroids fall out into the lumen of the intestine and in 14 to 20 days develop into adult tapeworms.

The clinical symptoms of hymenolepiasis include abdominal pains, often of a colicky nature, diarrheal stools with mucus and sometimes with traces of blood, loss of appetite, nausea, vomiting, loss of weight, headaches, weakness, irritability, epileptoid attacks. The dwarf tapeworm is encountered more frequently in children than in adults, and the condition is a more severe one in them.

All types of tapeworms are *diagnosed* on the basis of the demonstration of their ova in the feces; besides, taeniasis and diphylllobothriasis are also diagnosed by the discovery of the proglottids (segments) of the respective tapeworms in the stools.

Treatment of all types of tapeworm infestation. Dehelminthisation of tapeworm hosts is performed with male fern extract or with pumpkin seeds. Urinalysis must be done before beginning treatment. The patient should take nutritious, light and easily assimilated food for a day or two before treatment (white bread,

toast, broth, cereal soups, purée, milk, kefir, clabbered milk, cottage cheese, thin cereals cooked with milk, steamed forced-meat patties, boiled fresh fish, fruit juice starch puddings, grain beverages, tea); sugar is permitted. The evening before the first day of treatment the patient is given a glass of sweet tea or roasted grain beverage with toast and a saline purge (see treatment of Ascariasis). Castor oil should be avoided. In the morning of the day of treatment a purging enema is given, and (before the patient has had anything to eat) one-half to one glass of a cool 1 per cent solution of sodium bicarbonate; this solution should be taken in small portions over a period of half an hour. After this the male fern extract is administered; it is preferable to give the medicine in capsules to older children who are able to swallow them; children who cannot do so, or who are too young are given the medicine in cereal, honey, jam (30 g); it is recommended to give the fern extract (in capsules or in cereal) in divided doses three or four times over one to one and a half hours, washing it down with a one per cent solution of sodium bicarbonate ($1\frac{1}{2}$ -1 glass).

Male Fern Extract Doses

Age in years	Dose in grams
1	0.5
2	1.0
3	1.5
4	2.0
5-6	2-2.5
7-9	3-3.5
10-16	3.5-4.0
17 and older	4-5

Note: Children whom sodium bicarbonate causes to vomit may be given plain water instead.

Fern extract doses have been computed on the basis of 0.5 g/kg body weight, but no more than 5 g in all.

Thirty minutes after the ingestion of the last portion of the medicine the child is given a saline laxative if he took the extract with the soda solution; if no soda was taken the laxative is administered one hour after the last portion of the fern extract. An hour and a half or two after the laxative the child is given a light breakfast, independent of whether the laxative was effective or not. If no stool is passed three hours after the laxative was taken a warm water enema is given (two to four glasses of water at 32-34°C, the amount depending on the child's age).

If the worm is expelled without its scolex, another one to three enemas must be given.

It is recommended to treat possible nausea or vomiting during the fern extract therapy by keeping the child at rest in bed, applying warmth to the substernal region, and giving him mint drops and chips of ice to swallow. Fifteen to twenty minutes after the vomiting has ceased the medication may be resumed; if part of the fern extract has been ejected with the vomitus the prescribed dose that has already been prepared should not be increased.

In certain cases administration of the male fern extract evokes various manifestations: slow pulse, unconsciousness, convulsions, cessation of respiration. These symptoms are treated with hot-water bags, hot drinks, inhalation of solute ammonia, cardiac agents (caffeine, cordiamine), also adrenaline or ephedrine; for a day or two after medication a light diet is prescribed, as during preparation for treatment.

Contraindications to male fern extract therapy are decompensated heart diseases, ailments of the liver, gastrointestinal ulcers, febrile illnesses. Emaciated or severely anemic children should not be given this treatment; a course of therapy aimed at general improvement and against anemia should first be administered, and only afterwards is the fern extract permissible. Young girls should not be given the extract during menstrual periods.

If the fern extract is ineffective in expelling the tapeworms together with their scolices (the scolex is the head of these worms), then the treatment is repeated in two or three months if segments of the worms are again discovered in the feces. As the dwarf tapeworms exist in great numbers they are expelled in three cycles over intervals of 10 to 12 days; however, the daily dose of fern extract is lowered in these cases to 0.15 g per year of life. The course of fern extract treatment for hymenolepiasis may be repeated in two to three months.

In view of the direct danger of contamination the feces of hymenolepiasis patients must be scalded with boiling water (two parts of water to one part of fecal mass) and only then disposed of in the usual manner. Expelled tapeworms of all types must be burned.

Pumpkin seed treatment (varieties of *Curcubita pepo*). In the presence of contraindications to male fern extract pumpkin seed is used as a taeniafuge and general anthelmintic. Two days before treatment the child is put on a diet deprived of vegetables and fruits (to lessen the mass of roughage in the bowels), daily an enema is given in the morning and a saline purge in the evening. The

enema is administered on a fasting stomach, whether there was a bowel movement or not. The raw pumpkin seed is shelled (leaving the inner green coat). The amount of shelled seed given depends on the child's age.

Doses of Shelled Pumpkin Seed

Age in years	Dose in grams
1-3	50.0
3-5	75.0
5-7	100.0
7-10	125.0
10-12	150.0
12-14	200.0

The shelled seed is crushed (in small portions) in a mortar; after the last portion has been crushed the mortar is washed out with 50-60 ml of water, and this water is poured into the dish containing the seed; into this 50 to 100 g of honey, jam, or a thick syrup are mixed. The resultant mixture is given to the child on a fasting stomach (in bed); he should take it in divided portions over a period of one hour. Four hours later a cathartic is given, and half an hour afterwards an enema, even if the patient has passed a stool. Food may be given after the passage of the first stool evoked by either enema or cathartic.

DISEASES OF THE LIVER AND BILE DUCTS

The liver is an organ that performs numerous functions; it is one of the regulators of metabolism. Disorders of the liver lead to various metabolic disturbances (dysfunctions of protein, carbohydrate, fat and lipid, pigment metabolism).

Various infectious diseases (scarlet fever, diphtheria, measles, dysentery, sepsis) may cause the development of an inflammatory process in the liver (acute hepatitis) that gives rise to various degrees of liver dysfunction; in its turn this condition immediately tells on the general health of the child.

Many childhood diseases cause enlargement of the liver. Normally this organ is palpable only in children younger than 5-7 years of age, and it is never very firm; at more advanced ages any enlargement of the liver under the right costal rim is pathological.

Many acute inflammatory processes in the liver are attended by jaundice; this type of hepatitis may be associated with certain infectious diseases (typhoid, paratyphoid), in some cases they are of a septic nature. However, in some types of hepatitis the principal site of the disease is the liver itself as a reflection of a peculiar specific infection.

A disease of this type, characterised by its epidemic nature, has been described under the name of epidemic hepatitis (Botkin's disease).

EPIDEMIC HEPATITIS (BOTKIN'S DISEASE)

This disease is often observed in childhood. It occurs most often in the autumn and winter, and its incidence is most common between the ages of one and five years (over half of all cases).

Epidemic hepatitis is often diagnosed under the names of catarrhal jaundice, epidemic jaundice, viral jaundice, hepatosis, hepatosis-hepatitis, infectious hepatitis, parenchymal hepatitis, viral hepatitis. In Soviet medicine it is known as Botkin's disease or epidemic hepatitis. The famous Russian physician Sergei Botkin (second half of the nineteenth century) was the first to describe the disease and to note its infective nature; the name "Botkin's disease" was proposed by Dr. Kisel much later.

Etiology and epidemiology. Botkin's disease is caused by a filtrable virus demonstrable in the blood (less frequently in the urine) of patients. This virus has now been isolated and called virus A; its incubation period is between three to four weeks. A second virus—virus B—has also been isolated; this virus is the cause of serum hepatitis (homologous serum jaundice); it is transmissible with blood or serum during transfusions if the donor's blood contains it, and it can also be passed on by hypodermic needles and syringes. The incubation period of the serum hepatitis virus is between two to four months.

The virus of the Botkin disease is transmitted through feces, urine, duodenal contents and nose and throat discharge of people who have it. The most contagious time is the prejaundice period and the first few days of evident jaundice.

Diagnosis is difficult in the prejaundice period; the disease may be assumed if the child has been in constant contact with people who have the infection. In such cases suspicious symptoms are enlargement of the liver and dyspeptic disorders; in severe cases of developing Botkin's disease C.N.S. symptoms are often observed.

in children, particularly in very young ones (depression or excitement, meningeal symptoms).

Clinical findings. Common symptoms of the disease are fever, headaches, at times abdominal pains and vomiting; inflammations of the conjunctiva and sore throat; such a condition continues for 5-7 days, and then the whites of the eyes, the skin and oral mucosa become yellow.

The jaundice becomes darker every day, but its degree may vary considerably.

In a certain number of patients (10 to 15 per cent) jaundice may be accompanied by hemorrhagic eruptions on the skin (roseolous, petechial, urticarial).

In protracted cases of hepatitis various forms of angiectasis (enlargement of capillaries) are observed, so-called "vascula spiders". It is supposed that their appearance is connected with the development of cirrhosis of the liver.

Hemorrhages or petechiae are usually not observed, but nose-bleed may occur.

When the jaundice appears the fever falls and the general condition of the child improves. The urine becomes very dark, and a clearly positive reaction to bile pigments is demonstrable; the stools become a pale chalk-like colour. Urobilin is discovered in the urine, and the blood bilirubin level rises sharply; the blood sugar fluctuates considerably. The pulse slows down. By this time the liver has become enlarged, protruding from under the ribs to the width of two to four fingers, particularly its left edge; this area is painful during palpation.

In almost half of all cases the spleen is also enlarged.

After about ten days the jaundice decreases, the stools take on a normal colour, the liver and spleen return to ordinary dimensions, although the liver may remain enlarged and firm for several months, and even years.

The total duration of the disease is between three and five weeks; relapses occur frequently.

In severe forms of Botkin's disease the above symptoms may be accompanied by brain conditions (intense excitement, at times hallucinations) and meningeal symptoms.

The fever becomes intermittent, signs of cardiac weakness are observed (rapid irregular pulse, dulled sounds, expansion of the heart). The enlargement of the liver and spleen is more significant in severe cases of jaundice than in the ordinary forms of the disease. Complications are often observed: enlargement of the cervical lymph nodes, otitis, pneumonia.

The severe form of Botkin's disease usually terminates in recovery, but its conversion to *acute yellow atrophy* of the liver (toxic dystrophy) is possible; this condition is the final stage of severe jaundice, but its incidence in children is comparatively rare.

The clinical symptoms of yellow atrophy of the liver (as well as the symptoms of the severe form of hepatitis) are characterised by a rapid decrease in the dimensions of the liver; this organ often shrinks back under the ribs, pin-point hemorrhages appear on the skin of the face and trunk, urinalysis shows albumin; deep disturbances of carbohydrate and protein metabolism and chelemia (poisoning by bile acids) result in coma.

In lethal cases extremely clear histological demonstrations are obtained of degeneration and necrosis of the liver cells in the central part of the hepatic lobes and of subsequent formation of connective tissue (cirrhosis) and partial regeneration of the hepatic cells. This explains the deep changes that occur in protein, carbohydrate and pigment metabolisms; these changes, being results of the disease, are the cause of death, or, in lighter cases, of impairment of liver function in subsequent life.

Prognosis for epidemic hepatitis depends on its form; usually excellent for the lighter cases, while fatal terminations are observed in 5 to 7 per cent of the severe cases; frequently stable symptoms of cirrhosis of the liver remain. Prognosis is unfavourable for yellow atrophy of the liver.

Prophylaxis. All epidemic hepatitis patients must be registered (as in any other infectious disease) and the district epidemiologist informed.

Every case should be thoroughly investigated from the point of view of a possible epidemic, and the source of infection traced. Any injections (against measles), or any blood or blood product therapy made prior to the illness (within several months) must be taken into account.

Anybody who has Botkin's disease or is suspected of having it must be isolated for no less than three weeks from the appearance of the jaundice or one month from the onset of the illness. Hospitalised children may be dismissed no earlier than the specified periods of isolation if their clinical condition permits; after dismissal from the hospital the child should be registered at the nearest out-patient polyclinic for regular and prolonged observation.

Current and final disinfection is performed as for other intestinal infections; however, the stability of the virus of epidemic hepatitis must be taken into account.

Both children and adults who have been exposed to Botkin's disease are subject to medical observation for a period of 50 days from the moment of cessation of contact with epidemic hepatitis patients; particular attention must be paid to children who attend child-care institutions and to the personnel of such and of food-catering enterprises.

When a case of Botkin's disease occurs in the group of a child-care institution the sick child has been attending a quarantine is declared; no new children are admitted to this group for 50 days from the day the last patient was isolated.

Children who have been exposed to infectious hepatitis should be given injections of gamma globulin. The dose is computed on the basis of 0.15 ml/kg wt. In the presence of further incidences among the exposed children the gamma globulin injection is repeated in 30 days (the same dose).

To avoid the transmission of infection during intravenous and intramuscular injections or examinations connected with the withdrawal of blood both needles and syringes must be thoroughly sterilised each time after they have been used (boiling for no less than 30 minutes). The needles and blades used for puncturing the skin to obtain blood specimens must be thoroughly sterilised by flame or boiling each time after use, or destroyed. Chemical disinfectants are not reliable. Special instruments should be set aside for hepatitis patients (needles, syringes, steriliser).

Bed rest is essential throughout the entire period of the Botkin's disease. During the first days of the illness the diet of the patient is restricted: meat and eggs are forbidden, as well as too many fats. Later on the child may be given the food he usually gets: milk, meat no oftener than 2-3 times a week (boiled meat only), vegetables, fruits.

It is recommended to give 50-100 g of milk curds (cottage cheese) every day; this is not only a food of high caloric value, it is also a protein that has a favourable effect on the restoration of normal liver function.

Regular bowel evacuation must be attended to; constipation is treated with enemas or saline purges (Carlsbad or Glauber's salt, one half to one teaspoonful a day to half a glass of warm water).

It is also good to give the patient honey and a 5 per cent glucose solution three to four times a day in doses of 200-300 ml; in severe hepatitis cases intravenous injections of a 20 per cent solution of glucose, 10-20 ml once or twice a day, are advisable

Lowered cardiac activity calls for caffeine, metrazol (cardiazol, leptazol), or other cardiac stimulators; warmth is applied to the liver (hot-water bag) when it is painful.

Alleviation of pain and sometimes a rapid disappearance of the yellowness from the eye whites and skin may be effected by the withdrawal of bile through a duodenal sound or tube, particularly in the presence of pains or unpleasant sensations in the area of the gallbladder. An ordinary hot-water bag is used for decreasing abdominal pains. Vitamin C (ascorbic acid) is recommended in epidemic hepatitis (0.05 g three times a day).

A good effect is obtained by the administration of campolon, a liver preparation rich in B-complex vitamins and other substances beneficial to liver function. It is administered in intramuscular injections of 1 or 2 ml daily or on alternate days, five to ten injections in all. Some authors recommend "water shock" for combating intoxication associated with Botkin's disease (the patient is given one-half to one litre of water on a fasting stomach for several days).

Mineral water also has a good effect (Essentuki No. 17 or 4, Borzhomi, Slavyanskaya, Smirnovskaya), from a half to a third of a glassful before meals.

SPIROCHETAL JAUNDICE (WEIL'S AND VASILYEV'S DISEASE)

This disease is rare in childhood, and usually does not affect children younger than three years.

Spirochetal jaundice is caused by a spirochete of the *Leptospira* genus—*Leptospira icterohaemorrhagiae*—that circulates in the blood of the patient only the first four or five days of the illness, then disappears, and is found in the urine beginning with the 12th to 15th day. Its demonstration is quite difficult. In some cases it is possible to prove that the disease was caused by this spirochete only by injecting a guinea pig with the patient's urine.

It is universally considered that the spirochete is transmitted to man from infected rats; the latter contaminate water and soil, food products, and also other animals (dogs) with their urine. Infection may also be transmitted while bathing, either by ingestion of contaminated water, or by penetration through the mucous membranes of the eyes and nose. Rat bite is also a source.

The incubation period is from 1-2 days to 3-4 weeks (seldom).

Clinical findings. The disease begins with a prodromal period, during which the patient has headaches and is constantly fatigued. Pains in the extremities are observed, and an itching of the

soles and palms; in some cases nosebleed occurs. Later the temperature rises, feverish chills and general nervous excitability appear; a rash may erupt, sometimes morbiliform, sometimes with hemorrhagic tendencies, accompanied by photophobia. Spirochetal jaundice is, as a rule, attended by renal lesion of the nephritic type, the urine showing albumin, casts, hepatic epithelium cells and blood. In severe cases delirium and meningeal symptoms are observed (rigidity of the neck, Kernig's sign, red dermography (dermographia rubra, a condition in which the skin is peculiarly susceptible to irritation, red elevated lines being formed by tracing the fingernail or a blunt instrument over it). Jaundice appears between the 4th and 8th days of the disease. In some cases the spirochetal infection does not evoke jaundice nor affect the kidneys, and the disease resembles an acute infection with high fever and brain symptoms. In such untypical cases diagnosis is possible only on the basis of the demonstration of the spirochetes.

Prognosis depends on the severity of the affection of the body as a whole, and on the degree of renal and central nervous system involvement.

Treatment. The procedures are the same as for Botkin's disease; however, in spirochetal jaundice the general toxic symptoms are more vividly expressed, and the cardiovascular system is more deeply involved; therefore, cardiac preparations are in greater demand (camphor, caffeine). Immune equine serum (immunisation by pure cultures of *Leptospira icterohaemorrhagiae*) is also used; it is given in doses of 40 to 60 ml in the first days of the disease.

Prophylaxis: rat control, boiled drinking water, antiseptic disposal of patients' feces and urine.

CHOLECYSTITIS AND CHOLANGITIS

Cholecystitis and cholangitis (or angiocholitis) are diseases that affect not only adults, but children as well, and much more frequently than was thought formerly.

These disorders are almost always observed together; they compile, as it were, a single disease that should really be called angiocholecystitis, in which this or that part of the whole biliary system is prevalently involved.

In certain cases the abdominal pains often observed in childhood prove to be due to lesions of the biliary ducts and the gall-bladder.

The liver as an entire organ is undoubtedly involved to a greater or lesser degree when the biliary ducts are affected. Therefore, angiocholecystitis in childhood is often accompanied by liver enlargement.

Medical observations of children subject to periodic abdominal pains have shown that they often manifest clinical symptoms of hepatcholecystitis that are sometimes accompanied by jaundice. Such cases were usually diagnosed as epidemic hepatitis and the patients were hospitalised. However, in many such cases thorough investigation of the duodenal content resulted in the demonstration of *Giardia lamblia*, a protozoan parasite, sometimes present in large numbers, besides inflammatory elements (mucus, leukocytes).

Lambliasic hepatcholecystitis is not evidenced by such an acute onset as epidemic hepatitis. The patient has periodical abdominal pains and slight elevations of temperature; the aggravation and alleviation of the process is usually so mild that hospitalisation is not even considered. Often the periodic attacks of abdominal pain are diagnosed as being due to worms, while diarrhea is taken for a sign of gastroenteritis; a sudden onset of sharp pains and vomiting is diagnosed as appendicitis and the child is hospitalised for surgery.

An important moment in the diagnosis of this type of ailment, as of any other type of cholecystitis, is the localisation of pain on palpation of the gallbladder (in the substernal region under the right costal rim), and in certain cases pains that are aroused by tapping the liver area.

The decisive factor in diagnosing lambliasic hepatcholecystitis is, of course, the discovery of the *lamblia* in the duodenal content.

Inflammation of the biliary ducts and of the gallbladder in children may be caused by various infections, as in adults.

Angiocholecystitis may be of a catarrhal, ulcerative, or suppurative nature. A characteristic onset is elevation of temperature (to 38 or 39°) accompanied by the above-described pains; no *lamblia* are found in the duodenal content, but numerous leukocytes are usually present.

Treatment. Bed rest throughout the entire feverish period; a carbohydrate vegetable and fruit diet; duodenal sounding, duodenal lavage with magnesium; warmth to the liver; for intensive pain an anodyne is given (pantopon, belladonna). Good results are also obtained in the treatment of lambliasic hepatcholecystitis with quinacrine hydrochloride in doses as for malaria. The

very first course of treatment with this preparation causes a significant improvement, and the child may be dismissed from the hospital if his temperature is normal and he has no pains; the subsequent two courses are given at home.

THE GALLSTONE DISEASE

This disease is very rare among children. The symptoms are almost the same as in adults.

Treatment is also the same as for adults; however, surgery has almost never to be resorted to in children.

CIRRHOSIS OF THE LIVER

Any enlargement of the liver, particularly when it is accompanied by hardening, is a sign of pathology. An enlarged and firm liver, as we have already seen, is the result of various types of general infection. In the majority of cases the liver returns to normal upon recovery from the primary disease. But in some cases the liver remains enlarged and firm for a long time, becoming firmer and firmer, and growing larger instead of smaller. Usually this is accompanied by an enlargement of the spleen; the latter, too, becomes more solid. This condition is due to cirrhosis of the liver (diffuse fibrotic scarring), a condition characterised anatomically by connective tissue growth.

Cirrhosis of the liver may be a complication of Botkin's disease; some authors have even proposed that the cirrhotic form of epidemic hepatitis be classified as a peculiar form of the disease.

Cirrhosis of the liver is often connected with chronic infections, such as malaria, tuberculosis, or syphilis.

In *congenital syphilis cirrhosis of the liver* is not rare. It appears as a result of a significant growth of connective tissue in the liver caused by its diffused swelling associated with the swelling of the whole organ.

The clinical aspect of this type of cirrhosis is characterised by the presence of a large, firm liver, and an enlarged and quite solid spleen. Syphilitic cirrhosis is usually not associated with jaundice or ascites. Prognosis is bad.

Treatment is specific, as for syphilis.

Atrophic cirrhosis of the liver, a condition observed in adults as the result of chronic alcoholism, is a most rare incidence among children.

In this form of cirrhosis the liver is not much enlarged and is palpated with difficulty; the organ is quite firm, in some cases its surface is felt to be nodular. The spleen usually proves to be enlarged on palpation. This type of cirrhosis is often accompanied by ascites. Prognosis is bad.

Hypertrophic cirrhosis of the liver is observed in children more often than the previous forms of cirrhosis.

Some authors consider this form of cirrhosis to be due to inflammation of the hepatic parenchymal cells caused by an unknown infection.

Clinically the disease is manifested by a significant enlargement of the liver accompanied by protracted, intensive jaundice and frequent attacks of feverish chills; the spleen may also become quite enlarged. Hypertrophic cirrhosis of the liver does not cause hemostasis in the portal vein, and is not accompanied by ascites. The disease usually goes on for several years and its termination is fatal.

It should be noted that both of these forms of cirrhosis—the atrophic and hypertrophic forms—are not sharply divided; in both forms the pathologic process is due to some, possibly one and the same, cause that leads to degeneration of the hepatic parenchymal cells, and this is followed by secondary changes in the connective tissue; there can be transitory stages between these two forms.

DISEASES OF THE RESPIRATORY SYSTEM

In the pathology of childhood diseases of the respiratory system (in particular pneumonia) are second only to gastrointestinal disorders.

From 20 to 30 per cent of all children's diseases affect the respiratory system, occurring mostly before the child is two years old. The mortality rate of these diseases takes third place among others.

It is mostly infants who are subject to respiratory disorders. This is not so much due to the anatomic and physiologic features of very young children as to the immaturity of their central nervous systems, first of all of the cerebral cortex, the chief regulator of all vital processes in the body.

In studying respiratory diseases it is necessary to bear in mind that isolated lesions of one or another section of the system as a whole (the nose, trachea, bronchi, lungs) are rare. Usually all the respiratory organs are involved, one of them more acutely than the others.

The clinical aspect of lesions of separate sections of the respiratory system, from nose to lungs, is often a symptom of some generalised illness, usually of an infectious nature (influenza, measles, whooping cough).

THE COMMON COLD (ACUTE RHINITIS OR CORYZA)

Acute rhinitis or the *common cold* (also called *coryza*) is a most frequently observed ailment of childhood. In the great majority of cases it is the reflection or onset of some generalised infection (influenza, measles). Rhinitis may be caused by heat, chemical, or mechanical irritants (dust, soot, fine particles of coal), but this is rare in childhood. Acute rhinitis (running nose) caused by influenza is often the chief symptom of this infection.

Anatomically rhinitis is characterised by a swelling of the nasal mucosa and a mucoid suppurative discharge formed in different sections of the nose.

The *symptoms* of rhinitis, besides unpleasant sensations in the nose and discharge from it, are general indisposition, fever, obstruction to breathing through the nose; this makes it difficult for infants to suck and causes general restlessness.

Later a number of complications may arise as the ears and lower respiratory organs become involved (otitis, bronchitis, pneumonia).

Congenital syphilis and diphtheria are infections in which rhinitis is a specific factor. In infants persistent rhinitis accompanied by hemorrhagic discharge is one of the most important symptoms of congenital syphilis (see *Syphilis*). Nasal diphtheria is likewise characterised by the presence of a mucohemorrhagic nasal discharge and crusts, and of a specific membrane in the nares (see *Diphtheria*).

Prophylaxis and treatment. As acute rhinitis may be a symptom of some type of infection it calls for the institution of therapy of the corresponding infection, and also ordinary prophylaxis against diseases of the respiratory system (sufficient nutrition, sunlight, air). Adults with colds should not be permitted to care for young children unless they wear protective masks over nose and mouth. Nursing mothers with colds should also wear such masks. When a baby has a cold, and his nose is bubbly and stuffy due to abundant discharge and swelling of the nasal mucosa he may find it difficult to nurse at the breast or bottle; a solution of adrenalin instilled in the nose, followed by a solution of protargol, rivanol or penicillin often alleviates this condition.

Rp. Sol. Adrenalini hydrochlorici 1:10,000 10.0
DS. 3 drops into the nose 3 times a day
Rp. Sol. Protargoli 2% 10.0
DS. 3-5 drops into the nose twice a day
Rp. Rivanoli 1:1,000 10.0
DS. 3-5 drops into the nose twice a day
Rp. Penicillini 50,000 u
Aq. destill. 5.0
DS. 3-4 drops into the nose

Specific rhinitis (accompanying syphilis or diphtheria) requires special treatment.

Fetid rhinitis (ozena or rhinitis sicca) is characterised by an offensive odour from the nose, a constant formation of crusts, marked atrophy of the mucous membrane unaccompanied by ulceration of the membrane or caries of the nasal bones.

The origin of ozena is still not quite clear; some authors have assumed that it is caused by a specific bacillus, *Salmonella foetida* (formerly called *Coccobacillus foetidus ozaenae*) that can exist in the nose of dogs and be transmitted to man; however, this assumption is still doubtful.

The course of ozena is greatly influenced by external environment; bad living conditions and poor food higher its incidence. The improved well-being of the population and medical services in our country have lowered the number of people afflicted with the ailment, and the course of it has become more benign.

The onset of the disease occurs in childhood; the odour of ozena is so specifically offensive that the disease may be diagnosed by odour alone. The smell is stronger in the morning after sleep; when the crusts are removed it decreases significantly or even disappears for a time. The patient himself does not feel it, as the atrophy affects the olfactory area. Other types of fetid rhinitis are syphilitic ozena, tuberculous ozena, and ozena due to foreign bodies in the nasal passages. However, in these forms of ozena the mucosa is always abraded, while in true ozena this is never so.

Prognosis is not favourable, the disease often goes on for years, the fetid odour and formation of crusts decreasing or even disappearing in old age only.

Treatment. The crusts should be removed regularly by means of various softening agents, and the secretory function of the mucosa improved by irritants. Mineral oil is often employed for softening.

Rp. Ol. Vaselini 10.0
DS. 3 drops into the nose twice a day

A good irritant is Lugol's solution:

Rp. Jodi puri 0.1
Kalii jodati 1.0
Glycerini 30.0
MDS. 5 drops into the nose twice a day

At bedtime the mineral oil is instilled in the nostrils.

Besides symptomatic therapy good effects are also obtained by surgery that is aimed at narrowing the nasal cavities; the crusts cannot accumulate so easily in narrow cavities.

NOSEBLEED (EPISTAXIS)

Bleeding from the nose may be the result of either generalised or local causes. The first are infectious diseases that are often accompanied by nosebleed at the onset (typhoid, diphtheria, measles, influenza), or certain non-infectious diseases (leukemia, severe anemia, thrombocytopenic purpura, scurvy, heart trouble). In girls older than thirteen years nosebleed may be of the vicarious type, taking the place of menstruation. The highest incidence of nasal bleeding is observed in hemophilia. Local causes of nosebleed are trauma due to falls or blows, and, most commonly, to nose-picking, which results in abrasion of the mucosa of the lower part of the nasal septum (partition).

Treatment. Mild bleeding may be stopped by inserting a cotton plug into the nose and compressing the nostrils between the fingers for about ten minutes; the plug is left for 6-12 hours even if bleeding has stopped, and later a small amount of petrolatum is applied to the nasal mucosa.

A child should not lie down when his nose bleeds, as he might swallow a lot of blood, and this would cause vomiting. The child should be placed in a sitting position, his dress or shirt opened, and his head bent forward slightly. Warmth should be applied to the feet and cold compresses or ice packs to the forehead and bridge of the nose.

If these simple measures are of no avail it may be necessary to pack the front part of the nose for 24 hours with sterile gauze or with cotton moistened in hydrogen peroxide. The plug is cautiously removed by wetting it with hydrogen peroxide or normal saline solution. If nosebleed persists packing of the lateral part of the nose by means of a special instrument may become necessary.

Large losses of blood during nosebleed are compensated by subcutaneous injections of normal saline solution (300-500 ml); in certain cases blood transfusions are necessary. Further treatment depends on the primary disease.

FOREIGN BODIES IN THE NASAL PASSAGES

Foreign bodies in the nasal passages (*corpora aliena nazi*) are often observed in children, as the latter are apt to push various objects up their noses (buttons, beads, small nuts, sunflower seeds, fruit pits, peas).

These objects irritate the mucosa, sometimes abrading it and resulting in nosebleeds.

The foreign body first causes sneezing, tears, sometimes headache; these symptoms pass away and the only sign that remains is stuffiness of one nostril and discharge from that nostril. The incidence of foreign bodies in the nose is highest among young children.

Diagnosis is established by examination with a nasal speculum and by probing.

Treatment. The foreign body must be removed, otherwise various complications can arise (involvement of the nasal sinuses, of the lacrimal ducts, and even meningoencephalitis). The child is told to blow his nose, and if this does not eject the foreign body it is removed by means of a special dull hook.

Recovery after the removal of the foreign body is rapid.

DISEASES OF THE THROAT (TONSILLITIS, ANGINA)

Acute tonsillitis is an inflammation of the tonsils with sudden onset, fever, general symptoms and intense sore throat. It is an infectious disease that may be caused by different microbes (streptococci, staphylococci, diplococci). Tonsillitis usually attends various infectious diseases (influenza, scarlet fever, diphtheria). It rarely affects babies younger than nine months, owing to their immature tonsils.

The following forms of tonsillitis are observed:

Catarrhal tonsillitis (or catarrhal angina) is a form in which only a slight inflammatory swelling and redness of the tonsils are observed.

Follicular tonsillitis is also characterised by redness and swelling of the tonsils; besides this, single yellowish spots, pin-point to pea-sized, appear on the surface of the lymph nodules.

Lacunar tonsillitis is a form in which the tonsillar crypts are involved and their contents project as whitish or yellowish spots or films between the nodules.

Peritonsillar abscess (quinsy) is marked by the formation, after ordinary tonsillitis, of an abscess (phlegmon) in the peritonsillar area in the anterior part of the arch of the soft palate on one or the other side, accompanied by a general swelling and asymmetry of the soft palate.

The above division of tonsillitis is only conditional, as combined forms are commonly observed, when all the tonsillar elements are involved simultaneously.

Besides the local symptoms that accompany tonsillitis (swelling and hyperemia of the tonsils and soft palate, pain on swallowing, enlargement of the regional cervical lymph nodes) constitutional symptoms are also observed. They include headaches, pains in the joints and the entire body, elevation of temperature, sometimes as high as 40°, loss of appetite. Vomiting, particularly in young children, is frequently observed. The nasal discharge must always be examined for diphtheria bacilli.

Tonsillitis usually lasts from five to ten days; sometimes the disease is protracted by complications, such as tonsillar abscess or otitis.

Prophylaxis and treatment. Close contact with people who have sore throats should be avoided, as in any other droplet infection. A child with a sore throat who attends nursery, school or is an inmate of a children's home should be isolated.

Bed rest is recommended; older children are given solutions of boric acid, sodium bicarbonate (2 per cent), or rivanol (1:500, one teaspoonful to a glass of water) to gargle.

A good effect is obtained by white streptocide, doses 0.1 to 0.3 g (depending on age) 3-4 times a day for three to seven days.

Simanovsky-Rauchfuss tonsillitis (ulcerative-fibrinous angina) is not to be confused with the above-listed forms of tonsillitis. The latter may be caused by various types of microbes, while the Simanovsky-Rauchfuss form has a definite pathogen, or rather two pathogens that are always found together; they are a fusiform bacillus and a spirochete. This disease is often observed in children, sometimes as an epidemic. Its propagation is facilitated by general emaciation, anemia, syphilis, tuberculosis.

There are two clinical forms of this disease, the *fibrinous* and the *ulcerative-fibrinous* forms.

The *first* form is encountered less frequently, in about 10 per cent of cases, and it is often confused with diphtherial membranes. Usually only one tonsil is involved; it becomes covered with a greyish-white fibrinous exudate; in distinction from diphtheria no significant enlargement of the regional lymph nodes is observed, and general and local symptoms are not definitely expressed. The duration of the disease is approximately one week; the temperature is usually not high.

The *second* form is encountered much more frequently (up to 90 per cent of cases). Its onset is acute, with a high temperature and headache; several days later the surface of one of the tonsils becomes covered with a greyish-green or yellow fibrinous exudate that subsequently involves the entire tonsil and may spread to the adjacent soft palate. The tonsil soon ulcerates; crater-like depressions with dirty-grey floors and uneven edges are formed on the tonsil. The patient's breath becomes fetid.

The illness continues for seven to ten days; by this time the tonsil clears up. However, in emaciated and weak children the disease may be protracted to several weeks. The Simanovsky-Rauchfuss type of tonsillitis is diagnosed by the characteristic aspect of the fibrinous exudate (membrane) accompanied by ulceration, and the unilateral lesion and fetid odour from the mouth. Final confirmation of the diagnosis is obtained by a bacterioscopic examination of pharyngeal smears that reveal, as a rule, the characteristic fusiform bacillus and spirochete.

Treatment is similar to that employed for all forms of tonsillitis: gargling the throat and mouth with various disinfectant solutions (rivanol, potassium permanganate). Nothing is usually prescribed orally, and the course of the disease is benign if the process is not associated with some other ulcerative-necrotic process (stomatitis).

If the child is weak or emaciated his constitutional resistance should be built up by general measures (fish-liver oil, vitamin C, full-value diet).

INFLAMMATION OF THE MIDDLE EAR (OTITIS)

Inflammation of the middle ear, otitis media, is observed quite frequently in association with infectious diseases (particularly with measles, scarlet fever, influenza, pneumonia). Ordinary tonsillitis can often be complicated by otitis. Gastrointestinal diseases in infants are also often accompanied by otitis (toxic dyspepsia, dysentery). Otitis is diagnosed by pressing on the mastoid

process in back of the ear; the pressure evokes sharp pain, making the baby cry out loudly, and causing general restlessness. However, this is not always a reliable method, therefore, if otitis is suspected (fever, restlessness, sudden shrill cries, meningeal symptoms) the eardrum should be examined with a speculum (for redness, distortion or absence of the light reflex).

Inflammation of the middle ear usually runs a benign course, although relapses are common. However, the possible complications, antritis or mastoiditis, are serious. Their occurrence in infants may be obscure; therefore, protracted cases of otitis associated with general symptoms of intoxication call for surgical treatment (mastoidotomy or antrotomy) even in the absence of external signs of mastoiditis.

Treatment. In uncomplicated cases the application of warmth (compress to the ear, blue light irradiation); pus accumulating in the middle ear may cause the eardrum (the tympanic membrane) to bulge outwards; in this case myringotomy (incision of the tympanic membrane) is indicated. Slips of gauze are inserted into the ear for drainage if any discharge is noticed. Upon the appearance of signs of mastoiditis (swelling and reddening of the mastoid area, tenderness and pain when it is tapped and when the ear is pulled forward) cold (ice) should be applied in the back of the ear. Surgery is indicated if the symptoms of mastoiditis do not disappear in a day or two. Specific measures for otitis are sulfa drugs and antibiotics (penicillin, tetracycline). If a purulent process in the tympanic cavity is suspected or when symptoms of mastoiditis appear penicillin is administered.

DISEASES OF THE LARYNX

Acute laryngitis is an acute inflammation of the larynx with a particularly high incidence among young children.

Often the swelling of the tissues of the voice box (the larynx) and of the tissues above or below it narrows the air passages, causing laryngeal stenosis. Laryngitis accompanied by marked stenosis of the larynx is called *croup* (true or false [spasmodic] croups).

Etiology. Acute laryngitis mostly appears in the primary stages of some general infection influenza; thus its causative agents are the same bacteria that cause the given infection.

Pathologic anatomy. Laryngeal lesions, diffused or localised, and of various intensity.

Acute laryngitis may be divided into several anatomic groups:

Catarrhal laryngitis in which only reddening and swelling of the mucosa are observed;

Ulcerative laryngitis (see section on measles); a form characterised by reddening, swelling and ulceration of the mucosa, either diffused or localised in a certain part of the larynx (the arytenoid and subchondral areas, the posterior part of the true vocal folds).

Diphtheritic laryngitis (true croup), characterised by the formation of a fibrinous membrane on the mucosa (see section on diphtheria).

Clinical findings. The disease usually begins with a cold in the head, sometimes coughing is observed. In several hours, or in a day or two the temperature rises and a barking cough of various degrees of hoarseness appears.

Approximately in a week the cough softens and the child recovers rapidly. In children younger than two years the disease is often complicated by tracheobronchitis or even bronchopneumonia.

Acute laryngitis is observed at times in the form of false croup (laryngismus stridulis, spasmodic croup). It is mostly young children that are affected by it. The onset is very sudden, or the condition may begin with the mildest of coughs. Very soon after the beginning of the cough, the same evening or night, the child suddenly experiences difficulty in breathing, becomes quite hoarse, and has a fit of loud, shrill, barking cough ("crowing"); he may become pale, even cyanotic (signs of suffocation). Notwithstanding these frightening symptoms, recovery from false croup is very rapid, usually one to two hours, although relapses may occur the following two or three nights. However, croup associated with measles is often a protracted affair. The respiratory difficulties that accompany acute laryngitis may be very pronounced.

Differential diagnosis is based chiefly on the fact that false croup has a sudden onset, occurs mostly at night, and attains its highest peak immediately, while the stenosis of true (diphtheric) croup is preceded by a prolonged period of coughing, hoarseness, loss of voice (aphonia); its progress is gradual and no improvement is observed in the daytime.

In infants false croup should be differentiated from *laryngospasm* (spasmodic closure of the glottis) in *spasmophilia*, a condition with a very sudden onset. Other characteristic symptoms and signs of this illness should also be taken into account (increased nervous excitability, rickets).

Retropharyngeal abscesses can also cause respiratory difficulties; however, the type of respiration is different (gasping breath),

swallowing is difficult, and the patient's head is usually thrown back. Diagnosis of this condition is greatly facilitated by digital examination. Other possibilities that should also be remembered when breathing difficulties occur are the *inspiration of a foreign body*, and bronchial asthma. However, constriction of the larynx and loss of voice are never observed in these conditions, while bronchitic symptoms are always present (dry and moist crepitations).

Other diseases of the larynx, less often observed, are tuberculous ulcers on the vocal cords, syphilitic lesions of the larynx, secondary constriction of the larynx following edema caused by burns.

Prophylaxis and treatment. The treatment of simple acute laryngitis and false croup is not complicated: plenty of fresh air, a well-ventilated room. Abundant warm drinks are recommended, alkaline drinks (borzhomi or hot milk with soda); mustard plasters to the chest.

The child must be kept in bed while he runs a temperature, and given warm or hot baths daily (at 37 to 40°C) for five to ten minutes. Violent coughing may necessitate anesthetics (but not for infants!): codeine (single dose 0.001 g per year of life), Dover's powder (single dose 0.01 g per year of life). Besides this, relief is obtained in false croup by hot foot baths, with or without mustard (at 40°C) for ten to fifteen minutes, and by steam inhalations. (A kettle is kept boiling in the room, and the mother holds an umbrella over herself, the baby, and the kettle). If true diphtheric croup is suspected the child must be hospitalised and immediately injected with diphtheria antitoxin.

DISEASES OF THE BRONCHI, LUNGS AND PLEURA

The lungs, considered as an entire organ, include the air passages—the bronchi, ranging from the large ones down to the small bronchioles, the alveolar tissue (the air cells of the lungs), and the intermediate tissue in which the smaller bronchi, vessels and nerves are embedded; all these elements together are enveloped in a serous membrane, the pleura. The various parts of the lung (the bronchi, alveoli, intermediate tissue, and pleura) are rarely affected individually, so close are their interrelations; the prevalently involved part is responsible for the clinical aspect of the lung disease. If the bronchi are chiefly affected the disorder is diagnosed as *bronchitis*, when the alveoli and intermediate tissue

are the principal site of the lesion the disease is called *pneumonia*, and predominant affection of the pleura is called *pleurisy*, or *pleuritis*.

Moreover, diseases of the lungs cannot be considered as local pathologic processes; they are always constitutional diseases. Besides specific lung manifestations such as coughing and dyspnea, the general symptoms are elevated temperature, headaches, functional disorders of the cardiovascular system, etc.

The teachings of the Russian physiologist Pavlov and the investigations of his pupils K. Bykov and Y. Frolov have shown that dysfunctions of the lungs as a respiratory organ depend less on organic changes in this organ than on disturbances of the cortical-visceral links in the organism.

BRONCHITIS

Catarrh of the bronchi is understood as being a pathologic process localised in the bronchial mucosa, accompanied by the discharge of a mucous sputum.

Lesions of the bronchi are often associated with a similar catarrhal condition in the mucous membrane of the trachea (the windpipe), therefore, "tracheobronchitis" is a more suitable definition of the condition. When the smaller subdivisions of the bronchi, down to the bronchioles, are involved the clinical aspect of the disease approaches that of alveolar involvement; the capillary bronchitis that is usually described as a peculiar form of bronchitis—bronchiolitis, does not, at the height of its development, differ in any way from the bronchopneumonia of young children; no sharp line can be drawn between this form of bronchitis and pneumonia.

Etiology. Acute tracheobronchitis is particularly common in babies; in the majority of cases it is one of the important and constant syndromes of either acute (influenza, measles, whooping cough) or chronic infections (tuberculosis, syphilis). In other cases bronchitis may be a secondary symptom of various noninfectious diseases (bronchial asthma, burns of the windpipe, foreign bodies in the bronchi), although these diseases do not exclude the possibility of secondary infection of the respiratory tract (including the bronchi) by various types of pyogenic microbes (staphylococci, streptococci).

Pathologic anatomy. In acute catarrhal bronchitis the mucous membrane is swollen and red, sometimes with small hemorrhagic spots.

Histological examinations show that the epithelial cells lose their villi and swell up into goblet-like cells that exude mucus profusely.

The mucosa itself is dry at first, but it soon becomes covered with a mucous, mucoserous or mucopurulent exudate.

In the great majority of acute bronchitis cases changes are observed in the mucous membrane alone.

In more violent and prolonged catarrhs, when the bronchitis has already become a chronic ailment, the changes in the bronchi may have gone farther, causing infiltration into the muscular sheath (mesobronchitis) and a subsequent spreading to the connective tissue surrounding this sheath (peribronchitis). The incidence of the latter condition is particularly high in influenza, measles and whooping cough.

Clinical findings. In the majority of cases acute bronchitis is a primary manifestation of some infection (influenza, measles, whooping cough), therefore, its symptoms are closely related to the clinical aspect of the causative disease.

Usually the cough that appears at the onset of the disease in association with a rise in temperature is dry, no sputum being brought up. Catarrhal symptoms are observed in the larynx and pharynx (hyperemia), rhinitis is often present. Auscultation of the lungs at first evokes indefinitely localised dry rales; sometimes these sounds are concentrated in one lung or in a definite part of a lung. Later moist bubbling rales are heard (medium bubbling). Fine bubbling rales, particularly in infants, accompanied by asthmatic breathing, are often the first signs of alveolar involvement, of the onset of pneumonia.

Nothing pathologic is usually elicited by percussion of the lungs; it is only in cases of protracted bronchitis (associated with whooping cough, for instance) that pulmonary emphysema may be noted (bandbox sounds, a decrease of cardiac dullness as a larger part of the heart becomes covered by the emphysematous lung). Young children swallow their sputum, and it is often ejected with vomitus. Children older than nine years, as well as adults, cough up the sputum and expectorate it. Usually this sputum is mucous, viscid, yellowish-white, sometimes purulent; if coughing is violent it may contain blood. When no further complications set in the child runs a temperature for several days (depending on the primary illness), then his cough becomes milder, less frequent, disappears altogether, and he recovers. Sometimes bronchitis may be more protracted; this is connected with the duration of the primary disease and with domestic conditions, particularly when the child is deprived of fresh air and sunlight for prolonged periods because of fear of his catching cold. Such unfavourable factors may lead to the development of a number of complications and of secondary anemia; the latter in its turn affects the duration of any illness.

Prognosis is very good for acute bronchitis; in childhood the conversion to chronic forms is rare. Even in protracted cases un-

complicated by pneumonia complete recovery is often attained by proper care and improved domestic conditions.

Treatment. Bronchitis usually requires only fresh air, good ventilation of premises, opening windows or transoms for prolonged periods while the child is asleep, a well-balanced diet. Warm baths (38-39°C) are indicated, especially for infants. Mustard plasters and mustard packs are recommended.

All these procedures are aimed chiefly at making the child breathe deeper, to ventilate his lungs more completely; this is to some extent prophylaxis against the onset of pneumonia.

Cups are used for children rarely as their good effect is quite doubtful.

The symptomatic drugs administered are usually expectorants or, on the contrary, anesthetics to keep the cough down. However, children do quite well without expectorants, spitting up spontaneously and sufficiently. Only alkalis in the form of sodium bicarbonate or mineral water (Borzhomi, Izhevsk water, Essentuki) are absolutely indicated; these agents moisten the mucosa of the throat and relieve the unpleasant dryness that sometimes evokes violent attacks of agonising coughing. Sodium bicarbonate can also be prescribed in a mixture:

Rp. Natrii bicarbonici
Liq. Ammonii anisati aa 1.0
Aq. destill. 100.0
MDS. 5 to 10 ml every 2 hours

Narcotic drugs such as codeine, heroin, or Dover's powder are not used for young children. Older children may be given codeine (single dose 0.001 g per year of life) and Dover's powder (single dose 0.01 g per year of life):

Rp. Codeini phosphorici 0.01
Sacchari albi 0.1
M.f. pulv. D.t.d. N.10
S.1 powder 2-3 times a day (for a child of 9-10 years)
Rp. Pulv. Doveri 0.1
Natrii bicarbonici 0.12
M.f. pulv. D.t.d.N. 10
S. 1 powder 2-3 times a day
(for a child of 8-10 years)

Children afflicted with any protracted process in the respiratory tract should live out in the country for some time.

BRONCHIECTASIS (CHRONIC BRONCHIECTATIC PNEUMONIA)

Bronchiectasis is a Latin term meaning dilatation of the bronchi. The bronchial enlargement in this condition may be of two types: *cylindrical* bronchiectasis, a uniform dilatation of a bronchial tube or of its part, and *sacculated* bronchiectasis, irregular dilatations in sacs or pockets. The enlarged section of the bronchus is called bronchiectasis.

The condition may be a *congenital* one, when no alveoli have been formed on the terminal subdivisions of some bronchial branches and the pulmonary tissue consists of air tubes alone; these tubes may either be enlarged all along their lengths, or only their smallest branches are enlarged, forming sacs or pockets.

However, *acquired* bronchiectasis is much more common than the congenital form. It is usually the result of chronic inflammation in the lungs that induce an abundant formation of connective tissue in the bronchial walls and around them; the superfluous tissue stretches the bronchus and fixates it in such a dilated state, so that it cannot collapse. This is why bronchiectasis is classified by many authors as a specific pulmonary disease, chronic bronchiectatic pneumonia.

Bronchiectasis is preceded by prolonged diseases of the bronchi (chronic bronchitis), or is associated with diseases of the pleura and pulmonary tissue, when protracted inflammation has caused excessive formation of connective tissue that grows to the parietal pleura; this tissue is easily stretched by respiration and coughing, particularly if the adjacent bronchial stem has become involved. In such cases the bronchiectasis is usually of the saccular type.

The infections of childhood that most commonly precede bronchiectasis are measles, whooping cough and influenza; these diseases most frequently cause the changes in the muscular and peribronchial sheaths (meso- and peribronchitis) that ultimately lead to enlargement of the bronchi.

The chief *symptom* of bronchiectasis is a hacking cough with an abundant discharge of sputum, particularly in the morning; older children may expectorate very abundantly—by the mouthfuls.

The sputum is loose and purulent, with a strong, offensive odour. It often contains traces of blood, sometimes significant amounts of it.

Dyspnea is not ordinarily present without coughing; it appears only during the coughing attacks. Auscultation and percussion

often do not reveal any signs of changes in the pulmonary tissue, particularly in cases of cylindrical bronchiectasis; it is only when large sacs or pockets have been formed that auscultation and percussion elicit cavernous signs in some areas: amphoric respiration, tympanitic or dulled sounds, depending on whether the cavity contains air or fluid.

In not very marked cases bronchiectasis displays only the signs of bronchitis (dry and moist rales upon auscultation); a periodical discharge of abundant amounts of sputum, particularly if it is purulent and foul-smelling, may be the only symptom pointing to bronchiectasis.

Bronchiectases formed as complications of pleurisy (empyema) are sometimes accompanied by altered contours of the chest, a hollow forming over the area where the lesion was concentrated.

Another important symptom of bronchiectasis is the presence of so-called "drumstick fingers"—clubbing of the terminal digital joints (the toes may sometimes also become involved); this clubbing is chiefly due to hemostasis in the digital vessels, the result of impeded blood circulation in the pulmonary circuit.

X-ray examination is the most precise method for diagnosing bronchiectasis. For this purpose some roentgenopaque contrast medium is first introduced into the bronchi, as for gastrointestinal roentgenoscopy; this substance must merely coat the inner walls of the bronchi, leaving a space for the passage of air.

The substances used as contrast mediums are sprays of lipiodol or of 40 per cent iodopin; by means of this method it is possible to make a clear diagnosis of general dilatation of the bronchi and even to establish its character—cylindric or saccular.

The course of bronchiectasis is a prolonged one. At first the child's general health may not seem affected; his temperature rises only with complications and large accumulations of fluid in the enlarged bronchi. The most common complications are recurrent pneumonia; prolonged cases may develop into putrid bronchitis, gangrene of the lung, and amyloid degeneration.

Prognosis. Recovery occurs only in mild cases; complications being frequent, prognosis for bronchiectasis is always doubtful.

Treatment. Proper hygiene is of primary importance; as much time as possible should be spent in the fresh air, and in the summer the child should be taken out of town.

In order to facilitate the ejection of the sputum from the enlarged lungs, particularly in the morning, it is recommended to

place the patient on his stomach in bed, his head hanging down over the side of the bed; quite a large amount of fluid may be ejected in this manner.

Medicinal therapy includes preparations that decrease the secretion of sputum, and expectorants. They are administered in the periods between attacks. Antibiotic therapy is indicated for infection in the paranasal sinuses and the bronchi (a change of antibiotics is necessary from time to time as they alter the bacterial flora). Good results in cases of elevated temperatures are obtained with sulfa drugs and penicillin; aerosol treatment (inhalation of atomised particles) with penicillin is frequently used in cases where stasis of the purulent fluid in the bronchi leads to the appearance of characteristic symptoms of purulent processes (high temperature with significant remissions in the morning and evening).

Chronic bronchiectatic pneumonia, in cases of limited involvement of the lung accompanied by a progressive tendency of the process, calls for surgical removal of the involved lobes, before the development of amyloid or gangrene.

FOREIGN BODIES IN THE RESPIRATORY PASSAGES

The penetration of foreign bodies into the respiratory passages (the throat, trachea, bronchi) is no rare occurrence in childhood, particularly at the stage when babies begin to crawl over the floor and put anything they find into their mouths. However, it may happen at a more advanced age too: during meals if the child chokes over his food, and during play if he takes nails or other small objects into his mouth, or chews various seeds or ears of grain and like objects.

A thorough examination must be made of the past history of the case (anamnesis) before diagnosing the condition. The inhalation of the foreign body passes unnoticed sometimes, and it may be aspirated into the larynx, trachea, or bronchi. The appearance of the characteristic symptoms depends on the dimensions of the object and on its localisation.

Localisation in the larynx presents a clinical aspect of laryngitis; if the body is not a heavy one (e.g., a sunflower seed) it may be in constant movement, sometimes becoming compressed between the vocal cords, causing periodical attacks of choking; sudden death is quite possible in such cases. Sometimes the foreign body is expectorated during its movement back and forth, and recovery is immediate.

However, if the foreign body has been aspirated deeper and is lodged firmly in one of the bronchi it may cause aspiration pneumonia; one of the chief symptoms of this condition is weakened respiration on the corresponding side. Sometimes a putrid sputum is expectorated.

When a foreign body becomes infixed in the bronchi a bandbox sound is heard on percussion, and respiration weakens. If the inhaled object moves during inspiration and expiration a clicking sound is heard on auscultation.

Without thorough anamnesis it is often difficult to diagnose pneumonia upon its appearance.

If it is suspected that the inhaled object is of metal (a hook, nail, pin) an X-ray examination is necessary for precise diagnosis.

Treatment. When the child is examined by a laryngologist soon after he has inhaled the foreign body and the specialist sees that the object is lodged in the upper part of the respiratory tract, he removes it by means of a special curved forceps inserted between the vocal cords; as the forceps reaches the object its branches are diverged and the object is removed either manually or with a pincers. If the object has penetrated deep into the trachea or bronchus the only reliable method for its removal is tracheobronchoscopy, a method that is both diagnostic and therapeutic (a special instrument, a bronchoscope, is inserted into the larynx or into a tracheotomic wound for inspection, or for removal of the foreign body). When this operation is performed by a specialist the incidence of fatal terminations is significantly lowered.

If bronchioscopy is not available the only way of saving the child is tracheotomy (cutting into the trachea).

BRONCHIAL ASTHMA (ALLERGIC ASTHMA)

Bronchial asthma, or, as it is also called, allergic asthma, is characterised by recurring paroxysmal dyspnea, cough, wheezing, mucous sputum and a sense of constriction of the chest. At other times the patients are usually in seemingly good health.

Etiology. Allergic asthma is observed in various stages of childhood, typical cases have been described in six-week infants. However, its incidence is highest at ages between eight and twelve years, and more commonly so in children who have had pneumonia repeatedly. The prevalent factor in the origin of allergic asthma is a disturbance in the cortical activity of the brain with subsequent failures in the subcortical mechanisms regulating the functions of the visceral organs, including the bronchial muscles, causing a spasmodic contraction of the latter.

The condition is caused by the stimulation of the nerve endings (interoceptors) in the lungs by some irritant. These irritants may be ingestants (eggs, strawberries, fish, wheat, milk, or drugs), inhalants (pollen, dust of flowers, wool, hair, or feathers); it is also likely that the asthma may be caused by previous infection, or by a physical or intrinsic factor.

Usually bronchial asthma develops in children after a number of preceding catarrhal symptoms in the respiratory passages resulting in dysfunction of the mechanism of respiration.

The bronchial muscles do not only support the bronchi and prevent their collapse; they also play an important part in normal respiration, as do the muscles in the alveolar septums. The lumens of the bronchi are enlarged and narrowed by the periodic contraction of the bronchial muscles; this facilitates the delivery of atmospheric air to the lungs. The activity of the pulmonary musculature is controlled by the central nervous system; the disruption of the normal function of this musculature is neurogenous in origin.

It is possible that the asthmatic attack is preceded by a number of insidious disturbances of the respiratory function of the bronchial muscles caused by infections or by other agents that irritate the mucous membrane of the bronchi. Up to a certain point the central nervous system, namely the cerebral cortex, compensates such harmful stimuli in the walls of the bronchi; however (according to Ivan Pavlov) a time comes when the thus intensified regulating activity fatigues the nervous centres, and a collision of nervous processes occurs; the response is a paroxysm of bronchial asthma. This may lead to the fixation of a constant pathological conditioned reflex to stimulation of the bronchial mucosa by certain odours or foods if these odours or foods have periodically caused such irritation. And so, when the child grows up and becomes an adult, bronchial asthma may have already become a fixed condition in him.

Clinical findings. In older children the clinical aspect of asthma is similar to that observed in adults. Usually the attack is spontaneous, but often some slight catarrhal symptoms may precede it. The child suddenly awakens at night with severe breathing difficulties (dyspnea); his face is pale, while his lips and extremities may become cyanotic. The child strains to get more air, his inhalations are incomplete, his exhalations slower and longer. Sibilant wheezes may be heard at a distance, auscultation elicits musical rales everywhere, the chest is distended (expiratory dyspnea and inspiratory distension of the chest).

This severe condition continues for two to three hours, sometimes longer; the dyspnea decreases gradually, the patient begins coughing, discharges an abundant foamy sputum, the attack terminates and the child falls asleep. A similar attack may recur on the next day, often at the same hour.

A series of paroxysms constitute an asthmatic attack that may continue for several days, after which the patient feels well for some time, then the paroxysms recur again. The cough is usually accompanied by the expectoration of a thick, viscid sputum in

which Charcot-Leyden crystals, Curschmann's spirals and a large number of eosinophils are demonstrable under the microscope.

The clinical aspect of asthma is somewhat different in very young children. Usually the attacks are preceded by several days of slight coughing, sneezing, and nasal congestion (catarrhal symptoms); this is followed by the sudden onset (at night or in the daytime) of typical expiratory dyspnea and cyanosis, the child becomes very restless. Upon auscultation dry sibilant rales mixed with moist rales are heard. The paroxysm subsides very gradually, cough and sibilant rales being prominent for several days.

It is often difficult to diagnose asthma in small children, as bronchopneumonia also causes significant dyspnea in them. The character of the dyspnea typical for asthma should be considered (expiratory). Moreover, asthma is characterised by the absence of feverish spells, by a periodical nature of the attacks, and also by auscultative signs: absence of dulling of the percussive sounds, bronchial respiration and moist rales, prevalence of numerous dry musical rales.

Prognosis: In childhood asthma is curable, differing thus from the asthma of adults. Usually asthmatic attacks disappear at puberty, often even earlier (but in some cases they may continue throughout life).

Complications: Frequent severe attacks of asthma in children sometimes result in emphysema of the lungs, as in adults, but this happens very rarely in comparison with the latter, as the elastic pulmonary tissues are more resilient in children.

Treatment: Treatment during attacks consists mainly of the administration of substances that alleviate or arrest the paroxysm. Such substances are adrenaline (Sol. Adrenalini hydrochlorici 1:1,000, subcutaneous injections of 0.25-0.5 ml and more); instead of adrenaline ephedrine is used (0.001-0.006 with 0.2 g of sugar depending on age) orally for ten to fifteen days two or three times a day, or a 1 per cent solution is given subcutaneously during paroxysms. Besides this, in some subacute cases a good effect is obtained by preparations of belladonna (Extr. Belladonnae 0.001-0.01, depending on age).

Of late tissue therapy (after Filatov) has been used successfully for allergic asthma: autoclaved skin, or placenta, are placed under the skin by incision or are injected through a special syringe, or else injections of aloe are given.

The attack usually subsides without treatment; sudden death during paroxysms is rare. Proper hygienic measures, mental in-

fluences, fresh air, and if possible, keeping the child outdoors all the time are the most reliable agents for checking asthma. The institution of such a regimen for prolonged periods causes the attacks to subside.

Sometimes relief is obtained by the surgical removal of enlarged tonsils and adenoids, and also by nose therapy (rhinitis), as these are also often factors in the origin of respiratory disorders in children.

PNEUMONIA

Pneumonia or pneumonitis is a disease resulting from inflammation of the lungs. It is one of the foremost factors in the pathology of childhood, taking third place in the causes of mortality among infants.

Two types of pneumonia are differentiated: *croupous* or *lobar pneumonia*, and *bronchopneumonia* or *lobular pneumonia* (also called patchy or catarrhal pneumonia).

Lobar pneumonia is characterised by abrupt onset and rapid involvement of one or more lobes of the lungs accompanied by the formation of a fibrinous exudate in the alveoli. Lobar pneumonia follows a cyclic course and terminates by crisis after about a week.

This form of pneumonia is very rarely observed in infancy, and if it does occur it is quite atypical; at more advanced ages it is more common, displaying its most typical aspect within the age range of six to fourteen years.

Etiology and pathogenesis. The overwhelming majority of lobar pneumonias are caused by pneumococci (*Diplococcus pneumoniae*) of groups I, II, III and IV.

Most authors agree that the infection is spread along the respiratory tract and initially involves a respiratory bronchiole (bronchiolitis, peribronchiolitis). After this the development of the inflammatory process in the pulmonary tissue depends chiefly on the lymphatic system of the lungs. The lesion most often arises in the lower right lobe, then in the lower left and upper right lobes; the upper left lobe is affected least often. Such localisation is due to the topography of the lymphatic nodes; the nodes of the upper right and of both lower lobes are embedded deep in the pulmonary tissue, while the lymphatic nodes of the left lobe are out of the lung, on the aorta.

Pathologic anatomy. The pathologic changes evoked by lobar pneumonia in the anatomy of children do not differ essentially from what is observed in adults.

The same four stages are evident: congestion, red hepatisation, grey hepatisation and resolution.

The fibrinous exudate in the alveoli is not voided through the bronchi; it gradually resolves during the first 5-7 days without fever, and the affected lung returns to its normal anatomic aspect.

Clinical findings. The disease almost always begins abruptly with a high fever. In distinction from pneumonia in adults, the cardinal symptoms (chills, pain in a side, rusty sputum) are often absent in children, particularly very young ones.

Instead of complaining of pain in a side children often complain of abdominal pain (they are not able to localise their pains yet).

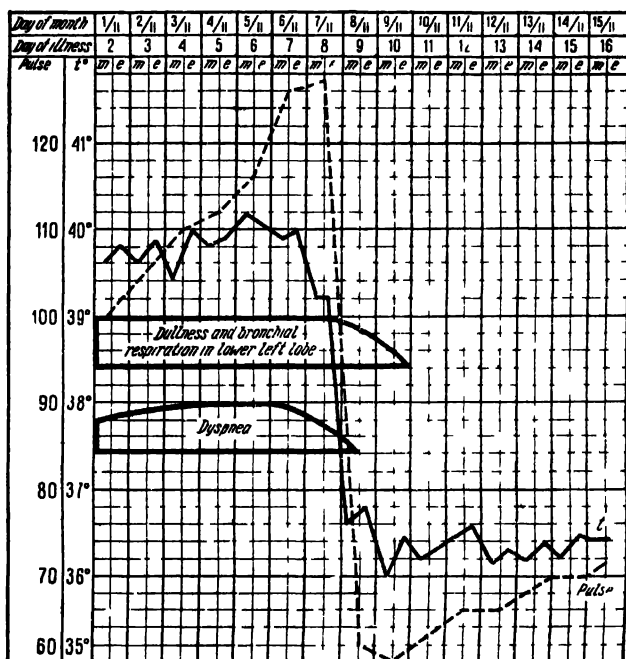


Fig 29 Lobar pneumonia in ten-year-old child (convalescence)

Children seldom expectorate sputum; it is only in older children that the characteristic rusty sputum may sometimes be observed. In young children the onset of the disease is often accompanied by brain symptoms (convulsions, vomiting, and meningeal symptoms).

The cough is at first very mild, in some cases it is absent altogether. Respiration is accelerated (40-80 per minute). The pulse-respiration ratio falls from 4-4.5:1 to 2-3:1. Respiration is shallow, severe dyspnea is present, and the auxiliary muscles become involved in the respiratory act, the nostrils dilated and the softest

spots on the chest (the jugular depression, the epigastric region) indrawn; in some cases a lag is noted in the respiration on the affected side of the chest.

During the first few days after the onset of the disease auscultation and percussion do not always elicit any pathologic signs; only on the 3d-5th day does any characteristic sign appear: first bronchophony, then bronchial respiration and dullness. At the height of the disease the temperature runs to 40°, nervous symptoms increase (delirium, unconsciousness). The decreased cardiac activity (dulled heart sounds, arrhythmia) usually observed in adults is rarely present in children. This type of pneumonia lasts one to nine days and ends with a *crisis* (Fig. 29). Improvement is very rapid, faster than in any other type of febrile disease; a child may be desperately ill one day, even unconscious, but may improve so rapidly after the crisis that he can sit up and play in bed next day.

Examination of the afflicted lung usually does not present such a sharp change: bronchial respiration and dullness remain, crepitations and fine bubbling rales are heard. However, complete resolution of the morbid process does occur in the lung, although somewhat later; the exudate completely resolves in the lung itself, the anatomic and functional characteristics of the lung become completely restored.

There may be deviations from the above-described typical pattern of lobar pneumonia, and in such cases it is easy to confuse the disease with some other ailment.

Thus, children often complain of pain in the right side of their abdomen, and this leads to diagnosis of *appendicitis*, the diagnosis becoming clear only after the characteristic symptoms appear in the lungs.

Moreover, the onset of lobar pneumonia is easily confused with *meningitis* in the presence of pronounced cerebral symptoms (vomiting, convulsions, semiconsciousness) and the appearance of meningeal symptoms (rigid neck, Kernig's sign and red dermography). In such cases spinal puncture is the only test for proper diagnosis.

In meningitis the spinal fluid is clouded, saturated with protein, and some type of bacteria is always found; if the disease is not meningitis the fluid is clear, contains no bacteria, and the protein level is normal. However, sometimes, particularly in infants, pneumonia may become complicated by true meningitis, and then the spinal fluid is changed, and pneumococci are found in it. Other common complications of pneumonia are purulent pleurisy (em-

pyema), nephritis, pyelitis, and epiphyseal osteomyelitis that is observed most frequently in infants.

In older children complications are rare, and prognosis is very good. The overall mortality rate among children of all ages is 1-2 per cent, while among children younger than two years it is 3 to 5 per cent.

Treatment. Lobar pneumonia follows a cyclic course that is seven to fourteen days long; therefore, treatment consists chiefly of bed rest in conditions most favourable for the natural course and termination of the disease. The most important agents are pure fresh air, good care, and nutritive, tasty, easily digested food (bouillon, milk, kefir or acidified milk, white bread); infants are given breast milk.

Lobar pneumonia responds very well to specific medication with sulfonamides and penicillin. As soon as the diagnosis has been established sulphamethazine, sulfathiazole, or some other sulfa drug is prescribed in doses of 0.2 g/kg/day, and penicillin 20,000 u/kg/day in three or four injections. Codeine is given for pains, and sedatives for poor sleep (luminal, bromural). The cardiovascular system is always affected to some extent by lobar pneumonia, but the most critical moments are directly before and during the crisis. Camphor and caffeine injections are the cardiacs of choice, particularly just before the crisis (signs: dulled sounds, weak irregular pulse, arrhythmia):

Rp. Ol. Camphorae 10% 10.0

Sterilis!

DS. 0.5 to 1.0 ml subcutaneously several times a day

Rp. Sol. Coffeini natrio-benzoici 1% 100.0

DS. 5 to 10 ml (1 teaspoonful or dessertspoonful)

2-3 times per os

For severe dyspnea mustard plasters and packs are indicated, for cyanosis—oxygen. Antipyretics are not administered, as high fever is the reaction of the organism to bacteria; it is only when the fever continues at a constant and very high level (40° and higher) that warm baths (at 37°C) or warm wet packs (36-37°C) twice a day are permissible. Diathermy of the lungs gives very good results. The child should be given vitamins C, A, and others from the very first day of the disease.

Bronchopneumonia. Lobular pneumonia is more common in very young children; its course is slower than the course of lobar pneumonia. The site of infection is local, involving only lobules, not the entire lobe of a lung. It is in very rare cases of so-called

fused pseudolobar pneumonia that an entire lobe, or even several lobes, are involved.

Etiology. The causative agent of bronchopneumonia, as of lobar pneumonia, is the *Diplococcus pneumoniae* (pneumococcus), chiefly of groups IV and X, less often the streptococcus and influenza bacillus.

Pathological anatomy. Microscopic examinations show that the volume of the sites of lesion is restricted by a lobule of the lungs, not by a whole lobe as in lobar pneumonia. Sections of the involved lung have a patchy aspect: the consolidated airless, yellowish-white or greyish-red sites of infections ranging from pea-size to nut-size are interspersed with normal air-conducting pulmonary tissue, emphysematous in places; in the spots where the bronchi are plugged there are atelectatic areas (collapsed airless areas). The exudate in the alveoli is usually not fibrinous (differing from what is observed in lobar pneumonia); it rather resembles the exudate attendant on various forms of inflammatory catarrh (giving rise to another name for this form of pneumonia); it is commonly serous, seropurulent, hemorrhagic or mixed.

In distinction from the exudate observed in pulmonary edema, this exudate contains many purulent elements, leukocytes and alveolar and bronchial epithelium.

Clinical findings. The course of bronchopneumonia differs in babies (younger than two years) and in older children.

In infants it usually takes an aspect of a generalised infection with local, often very slight, lesions in a lung.

In very young children the onset of the disease is frequently not associated with any preceding infection (influenza). In other cases anamnesis shows influenza, followed by laboured breathing, the general condition takes a sharp turn for the worse, and a clear clinical pattern of pneumonia develops.

The temperature is usually quite high (Fig. 30); however, in malnourished or atrophic children, or in children afflicted with a severe gastrointestinal disorder, pneumonia may develop at a subfebrile or even normal temperature; however, this should not be assumed to be a sign of a weak infection. Pneumonia in such children may be very severe even at normal temperatures, and its termination may be lethal.

In diagnosing pneumonia in young children it should be remembered that very often auscultation and percussion yield almost no results; quite often true bronchial respiration is absent, sometimes only bronchophony is noted on the involved side, frequently even weakened respiration.

At the onset of pneumonia the fine moist rales are often not auscultated.

During the progressive development of the disease all the symptoms of pneumonia are fully displayed. An early symptom

that should be sought is dyspnea (laboured breathing); it is expressed by dilation of the nostrils, and pulling in of the softest places on the chest (particularly in rachitic children), a wheezy grunting respiration, a cyanotic nose-lip triangle, emphysema.

In severe cases cyanosis and weakened cardiac activity are observed (dulled sounds, fast pulse, arrhythmia).

Lobular pneumonia in infancy follows a very diverse course, ranging from cases with a mild form accompanied by insignificant

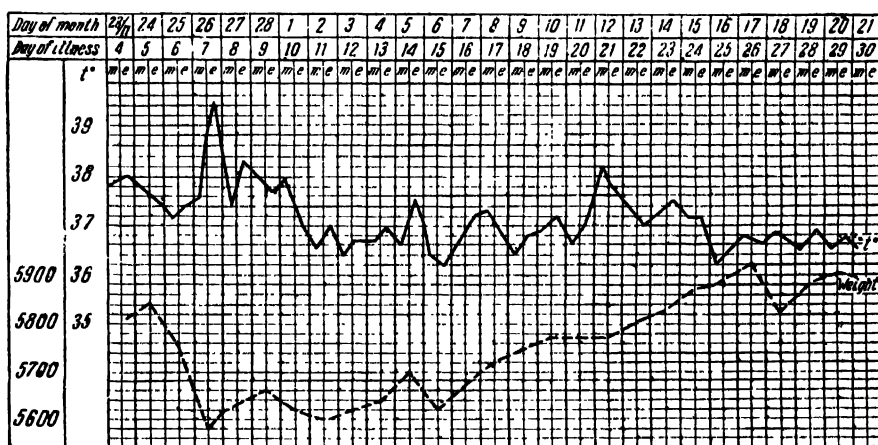


Fig. 30. Bronchopneumonia in five-and-a-half-month-old child

changes in the general state of the child to severe *toxic or septic* forms that are frequently followed by grave complications: meningitis, osteomyelitis, arthritis, otitis and antritis.

Severe nutritional disorders (toxic dyspepsia) complicated by pneumonia often have lethal terminations. This type of pneumonia differs somewhat in localisation: the usual pulmonary lesions associated with digestive disturbances are the lower posterior lobes of the lungs (paravertebral pneumonia); this is commonly due to the fact that congestion and atelectasis most easily occur in these sites in severely ill children. Often this type of pneumonia is attended by no rise of temperature, dyspnea is slight, crepitations are auscultated in the back in the lower lobes of the lungs.

There is one more form of lobular pneumonia that affects infants. Its aspect is that of a fused catarrh of the small bronchi (capillary bronchitis, bronchiolitis); it is accompanied by severe dyspnea, and numerous fine moist rales with no definite localisation are auscultated, in some cases all over the affected lung.

X-ray examination usually reveals small patches dispersed throughout the lung; such patches are sometimes found during postmortem examination.

Pneumonia in *newborn* and *premature* infants is also worth mentioning. This form of pneumonia often looks like a severe septic disease and frequently accompanies generalised sepsis. In these cases pneumonia is an infection conveyed by the blood; it can be of aerobic origin only in rare cases when infected amniotic fluid is aspirated into the lungs during delivery and aspiration pneumonia develops.

In premature infants fine, moist, low rales are sometimes heard in one or both lungs (usually in the lower lobes), no other symptoms (dyspnea, coughing, fever) of pneumonia being noted, and the general state of the infant being satisfactory. Such rales are very unstable, and not always heard.

These symptoms are all connected with pulmonary atelectasis that is often observed in immature infants, but these atelectases often develop into real pneumonia.

In children older than two years lobular pneumonia is commonly observed as a complication of some infection accompanied by catarrh of the respiratory tract (influenza, measles, whooping cough), rather than as an independent disease. These infections may also give rise to lobar pneumonia, but lobular or patchy pneumonia is more common.

Interstitial processes in the pulmonary tissue are characteristic of influenza, measles and whooping cough (mesoperibronchitis); the development of bronchiectasis of various degrees follows; in some cases the growth of the connective tissue is so pronounced that carnification of the lungs occurs. This type of pneumonia is of a chronic nature, resembling tuberculosis, and is sometimes confused with the latter.

Pneumonia associated with whooping cough usually develops during the convulsive period, the typical whooping, convulsive cough disappearing at the onset of pneumonia; subsequently, as soon as pneumonia resolves and the temperature falls the typical cough returns.

Pneumonia associated with influenza differs little in older children from that of adults; it may appear at the very onset of influenza, frequently at a later stage.

The temperature is usually not a high one; sometimes the feverish period is quite protracted (up to 3-4 weeks or more), and a gradual (lyterian) fall of fever is observed.

The pathological changes that take place in the lungs do not

disappear so rapidly as in lobar pneumonia; sometimes fine moist rales are auscultated for prolonged periods after the temperature has returned to normal and the patient's general state is fair.

Prognosis for all cases of lobular pneumonia is grave, in contrast to lobar pneumonia which is a more benign process with a very low mortality rate.

Lethality is much higher for lobular pneumonia, particularly in infants.

Prophylaxis and treatment. Observations have shown that pneumonia is most common among children living in unfavourable conditions (bad housing, frequent contact with influenza patients, poor nutrition).

An important factor in the prophylaxis of pneumonia is fighting such infectious diseases as influenza, measles and whooping cough, all of which are often complicated by pneumonia. In hospitals and in child-care institutions children who have contracted pneumonia must be isolated to avoid spreading the disease among the other children.

Proper management and nutrition play an important part in lowering child mortality due to pneumonia. The sick child must be provided with all possible conditions to facilitate a favourable outcome of the disease (nutrition, fresh air, good care). The child must be kept at bed rest on a high pillow; during the day his position must be changed frequently, and he should not be allowed to lie on his back for long. Babies sick with pneumonia should be picked up from time to time and carried around in the arms. The sickroom should be aired frequently; one should not be afraid of opening the transom or part of the window. In some cases it may prove expedient to keep pneumonia patients out-of-doors, warmly dressed and covered. This is particularly important in protracted chronic pneumonia associated with influenza, as prolonged deprivation of fresh air affects the general state of the patient unfavourably and leads to a still greater protraction of the disease.

The sick child should be given frequent drinks. Oxygen is indicated for infants at the onset of the disease and not only during the period of most pronounced cyanosis. It is recommended to treat lobular pneumonia, particularly in babies, with two antibiotics instead of only one (penicillin, streptomycin, biomycin, levomycetin [chloramphenicol], terramycin, tetracycline). Medication and physiotherapeutic methods of treatment are the same for both lobular and lobar pneumonia. Complications call for corresponding treatment, surgical or therapeutic. In purulent pleurisy the pus is aspirated from the pleural cavity by pleurocentesis; suppura-

tive otitis calls for special treatment. Prognosis for pyogenic meningitis is grave, but adequate early penicillin and sulfonamide therapy may lead to complete recovery; otherwise, prognosis for life and residua depend on the severity and duration of the disease. Weakened cardiac activity is treated with the cardiac preparations mentioned before. In some cases of pneumonia no response is obtained with sulfonamides, but albomycin (an antibiotic product of *Streptomyces subtropicus*) shows good results.

Protracted bronchopneumonia in babies responds to diathermy, hemotherapy, blood transfusion. A good effect is also obtained with hot baths (38-39°C), given for five to seven minutes daily.

PLEURISY

Lesions of the pleura, pleurisy or pleuritis, are commonly observed in childhood as secondary complications accompanying some pulmonary infection (pneumonia, tuberculosis).

According to the nature of the lesion pleurisy may be either dry or effusive (exudative).

Dry pleurisy is characterised by involvement of the pleural membrane without effusion of fluid; in most cases they are the result of tuberculosis. The pleura is also irritated and partially inflamed, without effusion, during pneumonia.

Older children affected with this form of pleurisy often complain of pain in one side of the chest, particularly when they cough hard. A characteristic *friction rub* is heard on auscultation over the affected area. The temperature is elevated. Prognosis is good for recovery; the pains subside, pleural friction rub sounds disappear, the temperature falls, and the child recovers. However, such a dry process is sometimes followed by the effusion of an exudate into the pleural cavity, and the aspect of the disease changes.

Exudative pleurisy in childhood may be of a serous (effusive) or purulent (suppurative) nature.

Serous and serofibrinous pleuritis are usually of a tuberculous origin, and only very rarely are they due to other causes (diplococci, rheumatic fever).

Purulent exudative pleurisy in childhood, particularly in infancy, is chiefly a complication formed during or after pneumonia. The causative agent is usually the pneumococcus (up to 30 per cent of all cases), less frequently the streptococcus, Pfeiffer's bacillus (*Hemophilus influenzae*), the staphylococcus and very rarely the tubercle bacillus. The incidence of suppurative pleurisy has become very low with the advent of antibiotics and sulfonamides.

Clinical findings. Purulent pleurisy, or empyema, is characterised by a secondary rise in temperature or remittent high fever (suppurative fever or pyemia) and dullness to percussion over the

affected area. The exudate may fill up the entire pleural cavity so that the lung is pushed upward as far as the first or second rib; this condition is usually attended by a widening and levelling of the intercostal spaces on the involved side, by *shifting of the mediastinal contents to the opposite side*, and sometimes by edema.

Breath sounds are decreased, while bronchial respiration is often heard over the dull area owing to the pressure exerted by the exudate on the lung.

A voluminous pleural effusion may accumulate in the pleural cavity during serous pleurisy (pleurisy with effusion); in children, particularly older ones, a percussive dullness is noted over a triangular area in the back on the healthy side of the chest (the paravertebral Rauchfuss-Grocco triangle), and an area of relative resonance in the low back near the spine on the involved side (Garland's triangle). One leg of the Rauchfuss-Grocco triangle, its base, is formed by the lower rim of the healthy lung, the other leg goes up the spine, its apex corresponding to the upper margin of the exudate, while the hypotenuse, a slightly curved line, descends to the outer angle of the base of the triangle.

Pleurocentesis (puncture and aspiration of fluid from the pleural cavity) is imperative for diagnosis on the slightest suspicion of pleurisy; pleurisy with effusion yields a purulent fluid in which the pyogenic agents are demonstrable. Empyema is not very difficult to diagnose, but in cases when the pus has accumulated only between the lobes of the lungs (*interlobar pleurisy*) the usual methods of diagnosis are ineffectual. The presence of interlobar pleurisy is suspected chiefly on the basis of prolonged remittent fever after pneumonia. Thoracocentesis (pleurocentesis) is of little use, as the puncturing needle does not always find the effusion; only X-ray examination can yield a clear picture of this form of pleurisy.

The same may be said of mediastinal pleurisy, a form in which the exudate accumulates in the cavity between the visceral pleura enveloping the lung and the pleura that lines the side of the mediastinum. This form is characterised by an area of dullness adjacent to the heart that is seen as a sharply defined shadow on X-ray photographs.

The course of purulent pleurisy depends on the volume of the exudate. A small amount of pus may sometimes resolve completely; in cases of significant effusions spontaneous recovery does occur, but adhesions often remain, as well as bands superimposed on the pleura. The adhesions are usually accompanied by a shrivelling of the lung, often by the formation of bronchiectases. Some-

times the pus breaks through into a bronchus and is coughed up with the sputum and thus voided.

Prognosis. The younger the child, the more severe the course of purulent pleurisy. Prognosis also depends on the nature of the pathogenic agent: pneumococcal pleurisy usually terminate more favourably than streptococcal pleurisy, as the latter are often of a septic nature and come to a rapid lethal termination.

Suppurative tuberculous pleurisy is rarely encountered in childhood; prognosis in these cases is very unfavourable. Interlobar pleurisy commonly follows a more favourable course even in very small children.

Treatment. The causative disease must be treated first. Dry (or fibrinous) pleurisy responds to warmth applied to the involved area (hot-water bags or electric pads); blue light is sometimes effective in alleviating pain in the side.

Analgesics (codeine, Dover's powder) are recommended for violent coughing and pains. Pus revealed by pleurocentesis should be withdrawn by repeated aspiration with ordinary syringes, or by means of a special apparatus, several times over intervals of three to ten days. If repeated aspiration of the effusion is not effective and its accumulation in the pleural cavity continues, while the child's general condition becomes aggravated, then thoracotomy and subsequent drainage are required. These local measures are associated with specific antibiotic treatment. Antibiotics are given intramuscularly (penicillin, streptomycin, albomycin) and orally (biomycin, terramycin, tetracycline).

It is likewise necessary to instill antibiotics into the pleural cavity (penicillin in single doses of 100,000 to 200,000 u).

Highly effective results are obtained with combinations of two or three antibiotics. Simultaneous administration of antibiotics and sulfonamides is also recommended (sulfodimezin, [sulfamethazine], norsulfazol [sulfathiazole]).

Moreover, proper care of the patient is very important; premises must be clean and well ventilated, food of proper dietary value.

DISORDERS OF THE HEART

The viability of the heart is much higher in childhood than in adulthood, as it has not yet been subjected to all the harmful influences that affect the adult heart (chronic alcoholic and nicotinic poisoning, overexertion, repeated infections). Besides, blood circulation is much faster in children, the lumens of the blood vessels

are wider, therefore, the cardiac musculature is nourished much better in children than in adults.

The cardiac rhythm and rate are subject to great variations throughout childhood, particularly between 12 and 14 years (arrhythmia). Excessive rapidity of the pulse, tachycardia is also not rare in children, becoming quite characteristic in certain infections (pneumonia, epidemic cerebrospinal meningitis, and scarlet fever). *Embryocardia* is often observed in the newborn as a *physiologic symptom* (the first and second heart sounds are almost identical). In babies and older children embryocardia is a morbid condition observed only in association with severe diseases (toxic dyspepsia, septic diseases, organic disorders of the heart).

The activity of the heart is closely related to the activity of all the other organs, therefore, many diseases frequently evoke functional disturbances in the cardiac activity of children. For the most part these disturbances are not accompanied by any gross changes in the muscles and valves of the heart, except during certain diseases (rheumatic fever, sepsis, diphtheria); usually the cardiovascular symptoms clear with recovery from the underlying disease.

The rate and rhythm of the heart are dependent on the condition of the central nervous system, on the regulating activity of the cerebral cortex and the related tonicity of the autonomic nervous system.

Various methods are employed for determining the functional capacities of a child's heart; this may be of particular interest after recovery from such a disease as rheumatic fever. Many of these methods are very complex and can be applied only in hospital surroundings. The simplest and most accessible method employed in routine practice for determining the functional capacity of the cardiovascular system is the respiratory test first proposed by a Russian physician, Shtange, and later somewhat modified.

The Shtange test is applied to children of school age. The child is seated on a chair and told to breathe in and out moderately, three times; on the fourth inhalation he stops breathing, pinching his nose (signal for noting time on stop-watch). When he cannot hold his breath any more he lets go of his nose (signal for end of test). Depending on age, a healthy child can hold his breath from 32 (at eight years) to 48 (at sixteen) seconds. Lesser figures point to functional insufficiency of the heart.

This test is also performed under stress; the child is told to squat five times in ten seconds, and the breath-holding time is measured directly afterward. The time will be somewhat lower under stress (22-25 seconds in school-age children).

The results of functional tests depend alike on the states of the heart muscle, the central nervous system, and the autonomic and endocrinic apparatus. When this test has been repeated several times the subject becomes accustomed to it and the results deviate from those previously obtained; this

should be taken into consideration. The respiratory test under stress is the more illustrative one; if the child is being kept at bed rest he is told to sit up and lie down several times (three, five, or ten times). Ambulant patients may be requested (besides the squatting exercises) to walk up ten steps, either slowly (in 20 to 30 seconds) or more rapidly (in ten seconds).

Heart trouble in childhood may be the result of *congenital anomalies*, or they may be *acquired* as a result of various generalised infections. Moreover, there are certain pathologic heart conditions of a *functional* nature (accompanied by no organic lesions of the heart).

CONGENITAL HEART DISEASE

Congenital lesions of the heart (inborn cardiac defects) are not infrequent. According to Dr. Gundobin their incidence comprises 0.5 per cent of all childhood diseases.

Congenital heart disease is caused by defective formation of the heart in fetal life owing to various harmful influences that affected the mother and the developing fetus during gestation.

Pathological anatomy. Postmortem examinations of children with congenital heart disease show, in the majority of cases, characteristic changes in the heart, resulting in typical lesions of the various areas of this organ.

Constriction of the pulmonary artery is most common; it is usually combined with the presence of an *opening* in the ventricular partition (*intraventricular septal defect*), or with persistent patent ductus arteriosus (Botallo's duct, which acts as a blood shunt between the pulmonary artery and the aorta in fetal life, does not close as it should after birth). Some types of congenital heart disease are due to inborn transpositions of the larger vessels, the aorta and pulmonary arteries changing places.

Numerous combinations of congenital heart defects are noted in postmortem examinations; many of these are not diagnosed during life, being discovered only on section.

Clinical findings. In many cases no typical clinical pattern is observable for the various congenital lesions of the heart, owing to their large number. It is only the most typical or clear-cut forms that display the more constant symptoms and signs of the disease. Congenital heart disease is characterised by certain *general* symptoms pointing to congenital heart failure without specifying the localisation of the lesion.

These *general* symptoms are:

In the very first years of life, and sometimes in the very first days, a systolic murmur is heard that cannot be explained by any acquired organic lesion of the heart or by any functional change in cardiac activity, particularly if the diagnosis is made at an early age, when functional murmurs cannot have been acquired yet.

However, cases of congenital heart disease have been registered when the murmurs are not heard for some time; it is only the seemingly causeless pallor of the child, and retardation of his growth and weight that lead to conclude that the so-called aphonic form of heart disease is present. The murmurs appear as the heart grows. Sometimes they weaken and disappear, to appear again later. During severe cases of pneumonia these murmurs may disappear owing to the weakening of the myocardium.

In children with aphonic heart failure even mild pneumonia follows a severe course often accompanied by sharp pallor and cyanosis.

Blueness (cyanosis) is sometimes noted immediately after birth even in the absence of heart murmurs; the blueness is most noticeable on the lips, oral mucosa, and extremities; it is the result of the mixing of arterial and venous blood, usually concomitant with constriction of the pulmonary artery. The blueness intensifies when the child cries or coughs (particularly in pneumonia). In some cases a sharp pallor is noted instead of cyanosis (pallid asphyxia); this pallor intensifies with certain diseases (pneumonia).

The general development of children with congenital heart disease usually tends to be retarded (low weight, small stature, cardiac infantilism).

Prolonged *polycythemia* (polyglobulia) may be present; this is a condition characterised by an increased number of red blood cells (6,000,000 to 9,000,000, and 110 to 130 per cent Hb) and by *macrocytosis* (the presence of macrocytes, or abnormally large erythrocytes, in the blood).

In some cases expansion of the heart is noted, usually to the right.

Very often the patient's fingers and toes are clubbed (drum-stick digits).

Sometimes the nails become curved in all directions, like a watch-glass (turtie-back nail).

In some cases a cardiac hump is observed, a bulging of the left side of the chest.

Prognosis. The majority of children with congenital heart disease succumb within the first years of life; only 10 per cent attain maturity. Prognosis is most favourable for isolated interventricular septal defect and for persistent patent ductus arteriosus. In all other forms of heart defects prognosis is grave; usually these children succumb to pneumonia and tuberculosis.

Children with congenital heart disease show poor tolerance of infections such as whooping cough; in some cases paroxysms of coughing lead to fainting spells, or even to death.

Treatment. Symptomatic. Compensated conditions call for proper regimen and diet only. Decompensation is treated with ordinary

cardiac preparations. A light diet is recommended, and restriction of fluid intake. The bowels must be evacuated daily. Long walks and physical exercises requiring strenuous exertion are forbidden; only light exercise in the fresh air is permitted. The child's room must be well ventilated. Overheating in the sun and fatigue should be avoided.

Surgical correction of certain congenital defects of the heart is of late becoming increasingly successful.

ACQUIRED HEART DISEASE

In children acquired heart lesions are due exclusively to some infectious disease.

Some acquired lesions are observed only during the infectious process, clearing up upon recovery; in other cases recovery is followed by the development of organic lesions of a more stable nature, lesions that remain for prolonged periods, or even for life.

Cardiac lesions in acute infections. Some phases of acute infectious diseases (scarlet fever, diphtheria, measles, typhus, typhoid, etc.) evoke significant changes in the cardiovascular system of the child.

Certain infections, as measles, epidemic cerebrospinal meningitis (meningococcus meningitis), affect the neuromuscular apparatus of the heart and its membranes; however, if they do cause some changes in cardiac activity these are mostly manifested as *functional* disturbances accompanied by no organic changes in the myocardium or endocardium. Usually cardiac activity returns to normal after recovery from the infection. Thus epidemic cerebrospinal meningitis is accompanied by a rapid pulse (tachycardia) from the very onset, but no changes in the myocardium or its membranes are observed either in life or after even in the most severe cases that sometimes end fatally.

Diphtheria has a more harmful effect, causing lesions of the myocardium, valves and coronary vessels. The most typical disturbances are noted in severe forms of diphtheria, but they may also accompany moderate and even mild forms of the disease if the antitoxin is administered late and/or in an insufficient dosage. These affections are of the nature of degenerative-inflammatory lesions of the myocardium. Heart trouble associated with *scarlet fever* (so-called scarlatinous heart) is only temporary, depending on the involvement of the cardiac nerve apparatus and on the change in the muscular tonus of the heart. Stable organic lesions

of the heart are not observed after scarlet fever; cases of endocarditis described as following scarlet fever are usually due to aggravation of a *rheumatic infection* that was present before the scarlet fever, and is to be looked upon as a fulmination of rheumatic endocarditis evoked by a new infection.

It is only septic scarlet fever that leads to the formation of *organic lesions* of the heart (septic endocarditis).

HEART LESIONS DUE TO SEPTIC DISEASE

Septic diseases of any etiology whatsoever (streptococcus, staphylococcus, diplococcus) may have sequelae in complications of the heart, all three layers (myocardium, endocardium, and pericardium) being involved.

Septic endocarditis (bacterial endocarditis) is characterised anatomically by ulceration of the valves accompanied by a deposition of fibrin; this type of endocarditis is of a fulminant nature. The *myocardium* and *pericardium* may become involved simultaneously with the endocardium or, in some case, separately.

Septic myocarditis does not differ essentially from myocarditis associated with an infectious disease.

Pericarditis secondary to septic diseases is characterised by purulent pericardial effusion. The clinical difference between purulent pericarditis and exudative serous pericarditis (due to tuberculosis or rheumatic fever) lies chiefly in the fact that purulent pericarditis has a more fulminant onset and violent course. The physical signs of an effusion in the pericardium (weakened heart sounds, area of cardiac dullness enlarged to percussion, and others) are the same as for exudative serous pericarditis.

HEART LESIONS DUE TO CHRONIC INFECTIONS

Such chronic infections as *tuberculosis*, *syphilis*, *rheumatic fever* may affect the heart in a manner similar to that observed in acute infections, immediately at their onset or during some periods of aggravation.

However, these infections are accompanied by other lesions of the cardiovascular system that are specific for the given infection (or at least more characteristic of it).

Tuberculosis in childhood is not accompanied by any particular lesions of the myocardium and endocardium.

In certain instances tuberculosis may affect the pericardium. Inflammation sets in on its outer surface, accompanied by a

sero-hemorrhagic effusion, and sometimes also by the eruption of small nodules.

This type of pericarditis may originate in infection spreading from neighbouring organs (the lungs, pleura, mediastinum, ribs), or the appearance of an effusion in the pericardium should be looked upon as being an allergic reaction to a tuberculous process in some organ, such as serous tuberculous pleurisy, and not necessarily direct transition of the infection from neighbouring structures. The incidence of tuberculous pericarditis is second only to rheumatic pericarditis; it is seen in 20 per cent of all cases of pericarditis of various etiology.

The clinical symptoms and signs of pericarditis are elevation of temperature, dyspnea, sometimes cyanosis. In the early stages a distinct friction sound is auscultated that disappears with the formation of the effusion, the cardiac impulse is barely palpable; the heart sounds become muffled, the cardiac dullness increases symmetrically in all directions. Sometimes the presence of a large effusion is manifested by bulging of the precordia (so-called heart hump). The pulse is very rapid, the first and second heart sounds are almost identical (pendulum pulse or embryocardia).

The child's general state becomes very poor, he loses weight, a generalised tuberculous process develops, and the child succumbs.

Chronic tuberculous pericarditis is a form characterised by an organising fibrinous exudate in the pericardial sac, on the mediastinum and pleura (tuberculous polyserositis). Often tuberculous pericarditis leads to the adhesion of the pericardial layers.

Heart disease associated with syphilis is much rarer in children than in adults.

However, syphilitic aortitis (independent of the localisation of the aortic lesion) are often discovered in life and on post-mortem examinations even in infants. The principal lesion is destruction of parts of the media as a result of syphilitic inflammation (mesaortitis); this often results in the formation of aortic aneurysms (dilatation of the aorta).

Malaria is usually not accompanied by any organic changes in the heart; it is only in severe cases of malignant malaria that symptoms of myocarditis are observed, as in any other severe infection. Commonly malaria is accompanied only by functional impairments of cardiac activity as a result of significant changes in blood constituents. (For rheumatic disorders of the heart see section on rheumatic fever.)

DISORDERS OF THE BLOOD AND HEMOPOIETIC ORGANS

CHILDHOOD ANEMIAS

Anemia is a term denoting a symptom, not a disease; it is observed in many childhood diseases, just as dyspepsia in small children is a symptom of many diseases that are chiefly manifested by gastrointestinal disorders. Both anemia and dyspepsia are particularly frequent symptoms that play a leading part in many diseases, therefore they are set forth in special chapters.

Although anemia is usually secondary to some disease, it may subsequently evoke all manner of complications and systemic disorders.

Sources of anemia in children are various disorders affecting the hemopoietic (blood-making) organs. Hemopoietic equilibrium is likely to be poorly established in children (particularly in young ones), bone marrow functions are still immature; these factors explain the frequency of anemias in childhood.

The *general symptoms* of anemias at all stages of childhood are pallor of skin and mucous membranes, but pallor may be present without any anemia; a pallid skin may be the result of vascular spasm. Blood tests are thus essential for diagnosing anemia. Characteristic signs are the reduction of the hemoglobin (Hb) to 60-40 per cent and even lower, and a reduction of the number of erythrocytes per cubic millimetre of blood (red blood count, RBC) from the normal 4,000,000-5,000,000 to 3,000,000-2,000,000. Sometimes the reduction of hemoglobin and red blood cells do not run together; while the Hb may fall considerably the RBC remains almost normal; this type of anemia is known as chloritic anemia or *chlorosis*. Besides these changes in Hb and RBC anemias of various origin and at various ages may be accompanied by changes in the types of blood cells circulating in the blood: often immature forms of both red and white cells appear in the peripheral blood. This is observed with particular frequency in early childhood. In some cases an insignificant factor may produce such a harmful effect on blood formation that numerous immature blood cells appear in the blood; it is as if they had emerged into circulation from the hemopoietic organs too early (nucleated erythrocytes, immature leukocytes and increased numbers of leukocytes).

Clinical findings. Poor appetite and general weakness, and sometimes fainting spells are symptoms common to all types of anemia. In older children anemia often leads to heart trouble (tachycardia, murmurs).

In studying the clinical picture of various forms of anemia it has been found convenient to divide anemias into two groups, differing one from the other in origin and course: 1) anemia of infancy and 2) anemia of older children.

Anemia of Infancy

Anemia of prematurity is a condition due to the functional immaturity of the hemopoietic organs connected with the premature birth of the child.

The symptoms of this form of anemia appear approximately by the sixth to tenth week of life. It subsequently passes away spontaneously without any treatment, but in certain cases it may persist for a prolonged period of time.

Anemias secondary to dietary deficiencies (*alimentary anemia*) are more commonly observed in the second half of the first year of life, between six and ten months. They are the result of monotonous diet, usually such a child has been getting only milk and nothing else at an age when children should already be having other food (fruit, vegetables). This type of anemia is more frequent in bottle-fed babies, but it is also often observed in babies who are exclusively breast-fed for too long.

Infantile pseudoleukemic anemia is a peculiar form of anemia observed in infants and young children. It is characterised by a severe waxy pallor; the hemoglobin level sometimes drops to as low as 20 per cent. The morphological constituents of the blood undergo both qualitative and quantitative changes: anisocytosis and poikilocytosis (the presence of red blood cells of various sizes and shapes). Blood smears often reveal nucleated immature forms of erythrocytes (normoblasts, megaloblasts). The red blood count drops to 2,000,000 or 3,000,000, while the white blood count rises to 15,000-20,000, single immature forms being noted among the white cells.

This form of anemia is observed in nutritional disturbances, chiefly in rickets. Besides the sharp pallor and peculiar blood pattern, this disorder is accompanied by enlargement of the liver, and, in particular, of the spleen; the latter often takes on the appearance of a large, firm tumour extending to the navel.

Such severe changes in the blood bespeak significant involvement of the seats of blood-formation that accompany the disease. However, this type of anemia often terminates with complete recovery.

Besides the above characteristic forms of anemia, there are forms that are encountered in association with many other diseases of infancy (erythroderma, septic diseases, infections, syphilis, tuberculosis); these forms of anemia display no special traits, as they are concomitant with the primary diseases and differ but little from anemias in children of the older age group.

Anemia in Older Children

In older children anemia is observed as a transient symptom associated with many diseases, but in some of the latter the anemia becomes stabilised and exerts a specific influence on the course and termination of the disease. These forms of anemia are:

Anemia secondary to rheumatic fever commonly accompanies all forms of rheumatic infection.

Anemia secondary to malaria develops at an early phase of the disease; sometimes it precedes the feverish stage; the total volume of red blood cells is decreased by one-fifth during the very first attacks. During protracted attacks blood production is inhibited, and this may lead to pernicious anemia.

Anemia secondary to syphilis, usually quite pronounced, is the result of the direct invasion of the hemopoietic organs by the spirochetes.

Anemia secondary to tuberculosis is moderate in the great majority of cases; in many TB patients it is of the chloritic type.

Anemia secondary to worm disease is particularly frequent when the patient is infested with the fish tapeworm *Diphyllobothrium latum*, becoming pernicious in some cases; other tapeworms (beef, pork) and hookworms (*Ancylostoma duodenale*), as well as other helminths may also produce sharply pronounced anemias.

Anemia secondary to certain endocrine disorders, as, for instance, myxedema, diabetes, may be very stable and pronounced from the onset to the termination of the disease.

Anemia secondary to significant blood loss after surgery and various types of trauma is sharply pronounced at its onset; as the patient's general condition improves the Hb and RBC levels rapidly return to normal, except in cases of extremely large losses, when transfusions are necessary in order to save the child.

Pernicious form. Anemias secondary to the above diseases (and sometimes anemia with no clear etiology) may take on a pernicious aspect approaching the severe progressive anemia observed in adults.

There is also another form of anemia in children, connected with poor living conditions, absence of sufficient fresh air, improper diet.

This anemia may rapidly disappear with normalisation of the child's surroundings and diet.

Prophylaxis and treatment of anemias. The prevention of the various forms of anemia is connected with their origin. It is important to provide young children with full-value food and manage their nutrition properly; too large quantities of cow's milk should be avoided, and solids, such as vegetables and fruits, and also fruit juices should be added at the proper time (at six months).

Children of school age should not be overexerted; this means that the school authorities must organise and manage the teaching process properly, institute physical exercises and sports, outings; in the summer months the children should attend outdoor groups or go to camp, weak children should be sent to special sanatoriums to recuperate.

Child welfare is promoted by deworming practices. An important issue in the treatment of various infectious diseases is proper nutrition and care.

The basic rule for treatment is determination of the source of anemia and its elimination.

The above measures are usually sufficient for the treatment of anemia in early childhood; when the child is given full-value food and kept out in the fresh air as long as possible his condition improves quite rapidly.

Prolonged and/or severe anemias in children of all ages, associated with a drop in both hemoglobin and RBC, require the administration of preparations that stimulate hemopoiesis and the formation of hemoglobin. The preparations recommended for this purpose are iron and arsenic salts (the latter usually for older children). When administering iron it must be remembered that only large doses are effective; the small doses formerly prescribed had very slight blood-building effects.

Of the existing variety of preparations the best effects are obtained with inorganic compounds of iron, owing to the fact that they split off active ions readily. Among them are reduced iron (ferrum reductum, or iron by hydrogen) and saccharated iron carbonate (ferri oxidum saccharatus, or soluble ferric oxide).

Iron dosage currently administered is much higher than some years ago; the most effective preparation is reduced iron. Even infants are given 0.3 to 0.5 g three or four times a day, while older children may be given as much as 3 g per day. Some authors

recommend washing down every iron powder with a solution of hydrochloric acid (Ac. muriatici diluti 1.0; Aq. destillatae 100.0 5-10 ml per dose), some recommend giving the iron in orange juice.

The tonic effect of *arsenic* on the hemopoietic organs is less reliable; arsenic is not prescribed for very young children. Children of an older age may be given potassium arsenite solution (Fowler's solution, or liquor potassii arsenitis) orally, 1/2 drop per year of life in two doses, or by subcutaneous injections of 0.25 per cent exsiccated sodium arsenate (sodii arsenas exsiccatus), beginning with two graduations of a one-gram syringe and ending with five or ten graduations (depending on age).

Good sources of dietary iron, used in the treatment of severe forms of anemia, are prunes and liver; liver is given both in its natural form, and in preparations (extracts: hepatocrin, liver extract); by intramuscular injections of 1 ml of campolon).

Infants are given 20 to 50 g of liver a day, beginning with smaller amounts, and gradually bringing it up to the required one.

Children from one to three years of age may have 100-150 and more grams of liver a day, older children are permitted 200-250 g. Infants are given cooked and sieved liver added to bouillon, cereal, vegetable purée; older children may be given stewed or fried liver, or liver paste.

The following recipes are recommended for preparing liver:

Liver-and-egg soup. 150 g of liver is minced with 15 g of fat, thoroughly mixed with one egg, 10 g of minced onion and 10 g of cooked vegetables. The entire mass is rubbed through a metal sieve into boiling bouillon, and the latter is then brought to a boil once more.

Fried liver. An onion is pan-fried in butter, then another 30-40 g of butter is added, and in this 200-250 g of thinly sliced liver is fried 10-12 minutes; the liver is turned from side to side while it is being fried. Salt, vinegar, and pepper are added to taste.

Hepatocrin is given orally. Its dose depends on the concentration of the solution, as inscribed on the label; this dose is easily computed on the base of the above-mentioned amounts of liver given at various ages.

The duration of treatment with liver and hepatocrin is one to one and a half months. Treatment is renewed if relapses occur. The most effective method of treating anemia is blood transfusion, 5-10 ml/kg wt into a scalp vein or an antecubital vein. Transfusions are repeated 4-5 times over intervals of five days (in severe anemia).

Blood transfusion is a life-saving measure in cases of severe anemia, particularly if associated with large traumatic or surgical losses of blood.

LEUKOSES (LEUKEMIAS) IN CHILDHOOD

Morbid conditions of the blood-making organs (the bone marrow and spleen) may be displayed in two basic forms: as *leukocytosis*, an increase in the leukocyte count, WBC, above the upper limits of normal, or as *leukopenia*, a decrease below the normal number of leukocytes in the peripheral blood.

Many diseases of childhood are accompanied by leukocytosis, and it is physiologic during digestion of food. A very significant WBC elevation is observed during many illnesses, up to 50,000 and even more instead of the normal 6,000-8,000. Severe leukocytosis is present, as a rule, in various suppurative diseases of the skin, bones, and internal organs, as well as in lung infections (pneumonia, pleurisy).

The WBC is usually elevated in such infections as diphtheria, scarlet fever, whooping cough, rheumatic fever, epidemic cerebrospinal meningitis, and many others.

However, in certain infections, and during the prodromal stage of many others, the opposite is observed, the number of leukocytes in the blood being reduced. Thus, leukopenia is an important diagnostic factor in measles, typhoid, malaria, rubella, and poliomyelitis, occurring at some phases of these diseases.

In many diseases both leukocytosis and leukopenia are only temporary, the blood values returning to normal upon recovery from the underlying disease. However, a deep involvement of the bone marrow and the lymphatics (in children as well as in adults) may result in their pronounced hyperplasia (increase in size of a tissue or organ owing to an increase in the number of cells); this, in its turn, leads to hyperfunction of these organs, and the number of leukocytes in the blood sometimes attains gigantic figures (up to 2,000,000).

This type of hyperleukocytosis as a stable and progressive symptom is the most characteristic feature of the white blood disease (generally spoken of as acute leukemia or leukocythemia). The cause of this disease is unknown; hyperplasia of the hemopoietic organs (the bone marrow) is responsible for the emergence into the blood stream of large numbers of mostly immature white blood cells.

However, the same disease may be observed in forms with no particular changes in the white blood count (normal, or even reduced), but with significant changes in the white blood cells themselves.

This type of disease is now known as *leukosis* rather than leukemia. Leukosis accompanied by an elevated WBC, attaining 2,000,000 and even more is called *leukemic leukosis*; forms with reduced white blood counts are comparatively rare and are called *leukopenic leukosis* (aleukemic or subleukemic leukemia of some authors). The leukopenic form of acute leukosis should be differentiated from the aleukemic form, when the bone marrow undergoes cellular changes typical of leukosis, while the peripheral blood shows a normal differential blood count.

Leukoses are differentiated according to the type of blood cells that prevail in the WBC. *Myeloid* or granulocytic leukosis (or *myelosis*) in which the predominant cell types belong to the granulocytic series, neutrophils being most numerous; *lymphoid* or lymphocytic leukosis (also called aleukemic lymphadenitis), in which the predominating cell type belongs to the lymphocytic series. These two forms of leukosis are two absolutely independent diseases that never convert into each other. Leukosis is usually observed in children only after the age of three, being very rare in infants.

Clinical findings. The onset of the disease is usually accompanied by an elevation of temperature, headache, loss of appetite, fatigue, sometimes vomiting—that is, by symptoms often observed in acute infections. Intense pallor is observed, the skin assuming a waxy hue, skin hemorrhages occur, ranging from minute petechiae to large spots and hematomas. The mucous membranes also become pale, tending to ulceration, bleeding and necrosis. Final diagnosis is determined on the basis of blood values.

Numerous leukocytes are observed in the blood, differential counts showing the predomination of myeloid leukosis in the majority of cases, when a large number of neutrophilic granulocytes (immature forms) are found. Leukosis in children differs from that of adults in being very acute, in some cases resembling sepsis (skin hemorrhages), with frequent necrotic processes in the throat. The course of the disease is from several weeks to six months and termination is lethal.

Leukosis is usually accompanied by a significant, at times gigantic, enlargement of the *spleen*, and by swelling of various groups of lymphatic nodes that often form a tumour (on the neck, in the armpit). The liver may likewise be enlarged. Sometimes the

blood picture of leukosis is accompanied by the formation of tumour-like periosteal growths, particularly on the skullbones, sternum and vertebral column; when these growths are localised in the orbital area protrusion of the eyeball is noticeable. The greenish colour of these growths has led to their being called *chloromas*; some pathologists consider that they constitute true tumours of the hemopoietic organs.

The aleukemic forms of leukosis accompanied by severe anemia sometimes follow a still more violent course in children.

Prognosis for both acute and chronic leukoses is always bad; in acute cases the lethal termination is very rapid (a month or two from the onset of the disease).

Treatment. No cure has been found for leukosis; treatment is directed at prolonging life and relieving symptoms. The most efficient methods, according to Soviet specialists are embichin (nitrogen mustard) therapy, sometimes in combination with local X-ray irradiation conducted under strict control of the blood picture. Some authors recommend vitamin B₁₂ in combination with corticotropin (ACTH), the cortisones (cortisone, hydrocortisone, prednisone), and the folic acid antagonists.

As a general measure blood transfusions (or erythrocyte mass transfusions) are given to control anemia, and antibiotics to prevent and treat infections (septic processes are often observed in association with leukosis).

INFECTIOUS MONONUCLEOSIS (IDIOPATHIC LYMPHADENITIS, GLANDULAR FEVER, FILATOV'S DISEASE)

A disease of the lymphatics was first described by Nil Filatov in 1885 under the name of "Idiopathic Lymphadenitis"; three years later the same disease was described as "Glandular Fever" by Emil Pfeiffer. Still later the disease became known as infectious mononucleosis.

The disease follows a course of an acute infection; the incubation period is between 5 and 18 days, but usually 5-8 days. The onset is abrupt, temperature rising to 39°, pains appear in the extremities, and catarrhal symptoms in the throat (redness and difficulty in swallowing); quite soon the cervical lymphatic nodes become swollen and tender, particularly along the posterior rim of the sternocleidomastoid muscle and on the back of the neck.

Enlargement of the liver and spleen are also noted. The patient runs a high temperature for eight to ten days.

The disease affects children and adolescents predominantly, but adults may also be affected.

Epidemic outbreaks of infectious mononucleosis have been described, although it is usually not very infective.

Etiology. The cause of infectious mononucleosis has not been finally determined yet. Some authors consider it to be a filtrable virus, others prefer

Listerella monocytogenes, a species of bacteria. The filtrable virus theory is supported by the fact that not only the blood of infectious mononucleosis patients, but also filtrates of this blood infect monkeys, in whom the clinical picture of the disease is similar to that of man.

Clinical findings. Besides the glandular form described by Filatov, two more forms of this disease have been described, the *anginous* form and the *feverish* (septic-infectious) form. All three forms have a common etiology. The division into three forms is conditional, as lesions of the glands, throat, and the presence of feverish chills are observed in all forms, but the degree of these symptoms varies. Sometimes the disease sets in with fever, and then lymphadenitis appears; in other cases a pronounced lymphadenitis appears together with the rise in temperature, and no correlation is observed between the intensity of the glandular swelling and the fever.

Other lymph nodes, besides the cervical, may swell and become tender: the axillary, submaxillary, inguinal, and retrocecal nodes. Enlargement of the latter nodes evokes painful sensations in the area of the cecum, so that it is sometimes diagnosed as appendicitis; cases have been described when such patients were mistakenly subjected to appendectomy.

The enlarged lymphatic nodes may attain the size of a hazel-nut or even of a walnut; in infectious mononucleosis the enlarged nodes are always painful, in distinction from painless glandular swellings observed in leukoses.

The swelling of the lymph nodes usually persists for ten to fifteen days, but it may continue for several months. Enlargement of the spleen is also a characteristic symptom; this organ becomes very firm and its dimensions return to normal only very gradually, long after the patient's temperature has dropped. The liver may likewise be enlarged, but not always.

Sore throat is observed in almost all cases; it may be of a catarrhal, lacunary, or ulcerative-necrotic type, resembling diphtherial angina; such patients are often hospitalised in diphtherial wards, although no diphtherial bacteriae are found in this angina.

The picture of the blood in infectious mononucleosis is quite characteristic; the true nature of the disease may be established quite rapidly on its basis. The WBC is not always increased, varying from very low figures (leukopenia) to significant numbers (hyperleukocytosis, 40,000 to 60,000). Cells of the lymphocytic and monocytic series are prevalent; high hemoglobin values and red blood counts are always observed at the height of the disease (Hb above 80 per cent and RBC not infrequently over 5,000,000). These values are somewhat reduced during convalescence.

No hemorrhagic syndrome is ever present in infectious mononucleosis. The absence of anemia and of hemorrhagic syndrome are an important feature in the diagnostic differentiation between glandular fever and true leukosis.

Prognosis is always favourable for complete recovery.

Treatment. Treatment is symptomatic, penicillin is indicated in the presence of ulcerative necrotic or diphtheroid angina; penicillin is less effective in the glandular form of the disease.

LYMPHOGRANULOMATOSIS (HODGKIN'S DISEASE)

This disease is rarely observed in childhood, and when it is it affects predominantly older children, boys oftener than girls. Its characteristic features are enlargement of the lymph nodes (chiefly on the neck and in the

armpits), sometimes to quite significant dimensions, pronounced anemia, and typical fever.

The etiology of the disease is still unknown. The majority of authors consider it to be an infectious (perhaps viral) hyperplastic process, others consider it to be a form of malignant tumour; there are more grounds for adhering to the first viewpoint than to the second.

Clinical findings. A characteristic symptom is the swelling of unfused lymph nodes, resembling, according to the figurative expression of A. Kisel, a "bag of potatoes". When it develops on the neck this "bag" may be of quite significant dimensions, larger on one side than on the other. The process highly resembles glandular tuberculosis, differing from the latter by the absence of fusions or fistulas and by a rapidly progressive course (fusions may appear later, but not fistulas). Biopsy or puncture of an affected lymph node are the decisive diagnostic factors.

The lymphatics become more and more involved, finally including the mediastinal and retroperitoneal lymph nodes. The liver and spleen become enlarged, progressive acute anemia is observed. The temperature curve shows intermittent hyperpyrexia, resembling typhoid fever; it may be quite protracted, with periods of remission.

Prognosis. Without treatment the disease goes on for 2-3 years, usually terminating lethally. However, of late treatment has been producing remissions of several years' duration.

Treatment. The most effective treatment currently employed by Soviet specialists is a combination of X-ray exposure and embichine (nitrogen mustard) therapy.

The affected lymph nodes and spleen (if affected) are irradiated daily, on alternate days, or over two-day intervals, depending on extent of involvement, 10 to 15 doses in all. Embichine No. 7 or neoembichine are the most effective nitrogen mustard preparations. The daily dose is 0.15 mg/kg wt intravenously in normal saline solution. X-ray therapy is administered first (in cases refractive to X-rays radium is given), and embichine after the lapse of one or one and a half months. The picture of the blood must be checked carefully, and embichine temporarily discontinued if the WBC falls as low as 3,000. Significant anemia and leukopenia are treated simultaneously with the other treatment by means of blood transfusions, cortisone, ACTH, vitamin B₁₂, and other substances stimulating hemopoiesis.

BLEEDING DISEASES IN CHILDHOOD

Many diseases of childhood are marked by various forms of hemorrhage (bleeding). These hemorrhages may be quite significant; they are symptomatic and typical in certain diseases (see *Epidemic Cerebrospinal Meningitis*). A hemorrhagic rash may appear in measles, smallpox (black smallpox), typhus, certain septic diseases, severe anemia, and others.

Such a tendency to bleeding may depend either on changes in the blood itself (reduced coagulation due to deficiency of fibrinogen in the blood), or on changes in the vascular wall; various infections produce toxins that affect the vascular walls and make

proliferation of the blood cells through it easier. Sometimes a grosser lesion of a vessel may be present (rupture). In some cases the bleeding may be due to a combination of causes.

Besides the infections known to tend to produce bleeding at some phase, there is a number of diseases of childhood in which hemorrhages are the leading factor; the etiology of these diseases remains unknown.

Idiopathic Thrombocytopenic Purpura (Werlhof's Disease, Essential Purpura)

This disease usually affects preschool and school-age children, girls more often than boys; it is sometimes of a familial type. Its cause is unknown, and it frequently appears without any preceding symptoms.

Clinical findings. A characteristic symptom is bleeding into the skin, and also into the subdermal tissue and muscles, the hemorrhagic spots ranging from petechiae to large bruises. The small hemorrhages are never symmetrical, and they often develop in places previously subjected to pressure (a fall or blow); nosebleed is likewise observed. The disease is usually accompanied by an elevation of temperature.

The following is observed in the blood: 1) bleeding time is prolonged; 2) sharply positive vascular symptoms are present (tourniquet, prick, hammer and pinch tests evoke hemorrhages in the skin); 3) clot retraction is absent or delayed; 4) thrombocytopenia is present (reduced number of thrombocytes), the thrombocyte count being 40,000-20,000 and even less.

The disease is a protracted one; at times there are remissions, when the bleeding seems to have stopped, but then it appears again, often in a worse form.

Prognosis depends on the duration of the disease. The acute anemia associated with chronic cases in which bleeding recurs constantly may lead to a lethal termination.

Treatment is symptomatic, bed rest being most essential throughout the hemorrhagic period. Hemotherapy is recommended (intramuscular injections of blood), in severe cases intravenous blood transfusions, vitamin K. Of late vitamin B₁₂ has been used for the possible anemia, with good results.

In severe cases, when bleeding is frequent and conservative therapy furthers no improvement a good effect may be attained by splenectomy (surgical removal of the spleen).

In the treatment of thrombocytopenic purpura an adequate and properly managed diet with a sufficient amount of vitamins (particularly vitamin C in fruit juices) is very important.

Nonthrombocytopenic Purpura (Schönlein-Henoch Syndrome)

This disease is more often observed in children of the preschool and school ages. It is also manifested by purpura; however, it differs from thrombocytopenic purpura by the presence of an exudative rash besides the hemorrhages.

At present the disease is looked upon as a hyperergic (hypersensitive) reaction of the organism to various infectious allergens (capillary toxicosis).

Clinical findings. Surface hemorrhages in the skin, in the form of minute petechiae, are characteristic; they are usually situated symmetrically on the body and extremities. But the most characteristic symptom of nonthrombocytopenic purpura, one that distinguishes it immediately from thrombocytopenic purpura, is the presence of spotty (sometimes vascular-spotty) exudative eruptions on the skin, besides the purpura. Often blood is discharged with the feces and urine (hematuria). Abdominal pains and joint pains (with swelling of the latter) are found in most children. Platelet count is not reduced, in most cases it is even elevated, a distinguishing feature in comparison with Werlhof's disease; at the same time eosinophilia may be present in some cases. The hemorrhagic tourniquet symptom is negative, bleeding time is not prolonged, coagulation (the opposite to hemophilia) is normal.

In nonthrombocytopenic purpura generalised toxic symptoms are also observed, such as malaise, headaches, loss of appetite, feverish state (often only subfebrile).

Prognosis for the ordinary form of the disease is almost always favourable.

The duration of the disease is four to eight weeks.

Occasionally the syndrome may be complicated by hemorrhagic nephritis, besides hematuria; in such cases the duration is several months, with occasional relapses.

Treatment. In mild cases bed rest, good hygiene, proper diet and vitamins (particularly vitamin C) are sufficient; in cases of severe hemorrhages and hematuria desensitising agents are given (dime-drol, or a 10 per cent solution of calcium chloride three times a day in doses of one teaspoonful, one dessertspoonful or one tablespoonful, depending on age). Blood and plasma transfusions are

given for intestinal hemorrhages. Of late the administration of ACTH has been recommended; it often brings the disease to a rapid termination. The ACTH dose is 10 to 40 units in 24 hours, depending on the age of the patient and severity of the disease. The preparation is injected intramuscularly four times within one day. Cases have been reported where a good effect has been attained in severe, relapsing forms of the disease by the administration of the Vishnevsky novocain blockade.

Hemophilia

Hemophilia is characterised by tendencies to severe hemorrhages after accidental injuries to the blood vessels; the bleeding is nowise proportional to its cause.

The disease is sex-linked and hereditary, occurs only in boys but is transmitted by women.

The symptoms of hemophilia are usually manifested in childhood, almost directly after birth; the appearance of initial symptoms at a mature age is extremely rare; on the contrary, in hemophilia patients the tendency to bleeding becomes less marked with age.

The disease becomes apparent the very first time the child's skin or mucosa are lacerated. Scratches, tongue-bites, blows, subcutaneous injections, tooth extraction are often the cause of lethal hemorrhages. Bleeding may occur into the joints, also into the subdermal tissue, muscles, and various organs.

Blood tests show neither quantitative nor qualitative changes in the morphological aspect of the blood, all its elements being normal; the abnormal feature is that *coagulation time is increased*, while the platelet count is normal.

Prognosis depends on the conditions in which the child lives, and on good care (to avoid accidental injuries). There are cases on record when a child succumbed to persistent postoperative bleeding as the physician was not warned of the fact that he had hemophilia.

Treatment. Symptomatic treatment is employed. During bleeding periods a good effect is obtained by *blood or plasma transfusion* (100-200 ml). Blood has an agglutinative effect owing to the high level of thrombokinase and the elements that produce it (thrombocytes, leukocytes) in the donor's blood. Less effective are subcutaneous and intramuscular injections of human and equine blood serum; they should be given only in cases where transfusion is impossible.

As in Werlhof's disease, the organism should be saturated with vitamins K and C. Fruit juices, dogrose hip infusions and extracts are recommended. Nosebleed calls for tamponing.

UROGENITAL DISEASES

DISEASES OF THE URINARY TRACT

Lesions of the kidney may be either of a functional character with no attending significant inflammatory processes or they may be conditioned by inflammatory or degenerative changes evident in various parts of this organ.

The kidneys are rich in blood vessels; the amount of blood circulating through them per unit of time equals to that passing through the lower extremities. From this large amount of blood the kidney produces a comparatively small amount of urine. The blood carries to the kidney the waste products from the whole body, from all the cells of the organism; owing to the large filtration surface of the renal glomeruli the blood quickly frees itself of the nitrogenous wastes of metabolism.

Various disturbances of this process (changes in the renal blood circulation with subsequent obliteration of the glomeruli under the influence of inflammatory processes) cause considerable changes in normal uropoiesis. The kidney starts excreting a smaller quantity of urine, excretion of the final products of the protein metabolism (urea) diminishes and a considerable amount of these products is retained in the organism; all this may lead to uremia (the presence of urinary constituents in the blood, and the toxic condition produced thereby). In addition, blood albumin begins to penetrate through the renal epithelium causing albuminuria (the presence of proteins in the urine), followed by edemas and dropsy (retention of fluid in the organism). The ureters may also be affected, their lesions being conditioned by the same infection which has caused renal disorder and closely connected with the affection of the kidney. There may be cases, however, when only separate parts of the urinary tract are affected (renal pelves, ureters, urinary bladder). In some cases there is a possibility of ascending infection due to the primary affection of the urinary bladder, ureters and renal pelves. Diseases of the urinary tract resulting from affection of one of its parts (kidney stones, deformation of the ureters, tumour, local tuberculous lesions) may also be observed.

Albuminuria in Children

Albuminuria in the newborn. Secretion of proteins with the urine (albuminuria) is still not infrequently observed in newborn babies. Some authors call it physiological albuminuria. As a rule, this kind of albuminuria does not last long (only a few days) and leaves no aftereffects. It is possible that the secretion of proteins results from congestions in the vascular system of the kidney sustained during labour.

Albuminuria in older children. Albuminuria is sometimes observed in older children after physical strain, excitement or cold baths, but it is of no pathological significance, the excretion of protein with the urine being negligible and temporary.

The presence of protein in the urine may be observed in many infectious diseases denoting a disturbance in the function of a kidney as a result either of congestive phenomena in it or of slight inflammatory affections of renal tissue. This form of albuminuria usually disappears with the symptoms of the main disease.

Orthostatic albuminuria. In children 7-14 years of age prolonged and stable albuminuria is sometimes observed; in this case the protein is not found in the morning urine after the night's sleep but is observed in considerable quantities in the daytime and evening urine, after the child has walked about. This form of albuminuria is called orthostatic, i.e., albuminuria which appears when the patient is in an upright posture. Most often it is observed in weak, pale and thin children who are not infrequently affected with functional cardiac disorders. The etiology of this form of albuminuria is unclear. It is supposed to be due to disturbance in renal blood circulation, which is particularly pronounced when the child is in an upright posture or has lordosis (forward curvature of the lumbar spine), which often leads to venous congestion. Orthostatic albuminuria produces no other symptoms save the presence of protein in the urine, does not lead to any renal diseases and passes without leaving a trace. No treatment is usually required: there is no need to keep children in a supine position or prescribe a diet as it is done in cases of patients suffering from renal diseases. Only the usual generally strengthening treatment necessary for a weak or nervous child—fresh air, proper food—is recommended; physical exercises and sports are not contraindicated.

Inflammation of the Kidney (Nephritis)

Etiology. Acute inflammation of the kidneys—nephritis—is not infrequently observed in children; it is mainly a result of various infectious diseases (scarlet fever, influenza, pneumonia and septic diseases) in which inflammation of the kidneys is one of the most serious complications. In some cases inflammation of the kidneys may appear suddenly, as a primary disorder, thus giving grounds

for assuming a primary infection of the kidney. However, even in such cases the renal affection was apparently preceded by some unnoticed infectious disease.

Inflammation of the kidneys may also be the result of some intoxication of the organism: intoxication by metals (mercury) or poisons (phosphorus, arsenic, etc.), but such etiological factors are less significant than infections.

Pathological anatomy. Anatomically, the affections of the kidneys may vary with the part of the organ that is affected the most. In some cases, as in scarlet fever, the inflammation primarily affects the glomeruli (glomerulonephritis). Histological examination reveals proliferation of the nuclei of the vascular endothelium, exudation and accumulation of microcellular elements in the glomeruli. In other cases, the disease may, in addition to the aforementioned affections of the glomeruli, also involve the renal epithelium, especially the epithelium of the convoluted tubules in which degenerative changes predominate; focal or diffuse degeneration of the renal epithelium is known as *nephrosis*.

Various infections, septic infections in particular, may also be attended with anatomical changes in the interstitial tissue of the kidney; these changes are characterised by microcellular infiltration which may be replaced by a scar in case of the patient's recovery. It should be noted, however, that anatomically the disease rarely affects only one of the aforementioned parts of the renal tissue; in the majority of cases combined lesions are observed. But even in such cases one of the lesions comes to the fore more often in the beginning of the disease, thus characterising one of the clinical forms of the disease and its course.

Clinical findings. Scarlet fever is the most frequent cause of *glomerulonephritis*, i.e., inflammation of the kidneys, in which the glomeruli are primarily affected. In the terminal stage of the disease the inflammation also affects the renal tubules (symptoms of *nephrosis*) and in grave septic cases may develop into septic *interstitial nephritis*.

Scarlatinal nephritis. Glomerulonephritis. In cases of scarlet fever nephritis usually develops as a complication in the third or fourth week of the disease; the temperature rises to 38-39° and higher, the child's face becomes pale and puffy, he is nauseated and sometimes vomits; frequent and tense pulse, accentuation of the second sound on the aorta and elevated blood pressure are observed.

The daily urinary output decreases sharply, the urine turning dark, sometimes red; albumin and erythrocytes, sometimes in great numbers, appear in the urine. These symptoms characterise an affection of the entire vascular system, especially the renal vessels, mainly the glomerular capillaries, and are the clinical manifestations of primary *glomerulonephritis*: as the disease progresses edemas develop, ascites may appear, the urinary output continues to

decrease and complete anuria is sometimes observed; the amount of albumin in the urine increases, large numbers of casts are observed in the urinary sediment. Such development of the disease characterises the secondary lesion of the renal tubules and is a clinical manifestation of nephrosis.

The disease may subsequently develop in two ways: all symptoms of the nephritis begin to subside; edemas decrease, the amount of albumin in the urine diminishes and the blood disappears and the child recovers or, simultaneously with symptoms of advanced anuria and the generally grave condition of the patient, the edemas increase rapidly and a picture of *uremia* develops.

As a rule, uremic eclampsia is observed. Clonic spasms develop at the height of the edemas; these symptoms are not infrequently preceded by headaches, nausea, vomiting, dilatation of the pupils, high tendon reflexes. The child often dies during the attacks with symptoms of cardiovascular and respiratory disorders.

It should be noted that in children uremia attending nephritis is rarely true uremia resulting from retention of nitrogen in the blood. Most frequently it is apparently due to retention of sodium chloride, cerebral edema and increased intracranial pressure.

Scarlatinal nephritis lasts an average of 4-6 weeks; not infrequently it produces relapses which, however, run a rather favourable course.

Death may be caused by uremia, ascites, pneumonia, empyema and skin affections (erysipelas).

Other infectious diseases—acute and chronic (influenza, pneumonia, diphtheria, tuberculosis, syphilis, chronic pyosis, etc.)—may also affect the kidneys, but these diseases are primarily characterised by lesions of the renal tubules, i.e., nephrosis. A clinical manifestation of nephrosis is the presence of a large amount of albumin in the urine; casts and cells of renal epithelium are found in the urinary sediment; erythrocytes are absent or rare.

Marked retention of liquid in the organism—edemas and accumulation of water in the cavities (ascites)—is sometimes observed. Nephrosis may be acute or chronic. Acute nephrosis usually runs a favourable course and frequently ends in recovery, while the prognosis of chronic nephrosis is often very poor (amyloid, lipid and mixed nephrosis).

Amyloidosis of the kidney. Amyloid degeneration of kidneys is not very often observed in children, especially very young children. It is more frequent in children past the age of ten.

Amyloidosis of the kidney is associated with various types of prolonged suppurative processes, tuberculosis and syphilis.

Anatomically this affection is characterised by special degeneration of the renal tissue which produces a peculiar colour reaction upon painting the affected part with iodine one drop of which stains the tissue a brownish-red, while intact tissue stains yellow.

The presence of prolonged pyrosis and cachexia, appearance of waxy pallor, easy fatigability and inappetence in children warrant suspicion of incipient nephrosis. The urine is usually light-coloured, but its output is unaffected; it contains a large amount of protein and there are separate granular and hyaline casts in its sediment. The symptoms include edema of the legs, ascites, and enlarged liver and spleen; as a rule, the disease ends lethally. Treatment instituted in due time may sometimes lead to recovery only in cases of syphilis.

Treatment. The treatment of inflamed kidneys consists mainly in sparing the affected organs, for which purpose the patient is prescribed complete rest and confinement to bed as soon as the disease has been diagnosed. During the first day the patient is kept on a starvation diet, only a teaspoonful of water being given to the child to quench the thirst. From the second day on the amount of liquid, usually tea with milk, is increased (300, 500, 600 ml). In addition the patient is given coffee, jelly, salt-free bread and fruit. The amount of sugar is increased with each day and on the third day it reaches 50-100 g. The child is kept on this diet for two or three days and eventually, if the urine output increases and edemas subside, the consumption of liquid is not limited and the diet is enriched with carbohydrates, fats, vegetables and lactalbumins (sour milk, cottage cheese). Later eggs may be prescribed, and in terminal stages of the disease meat (boiled) may be added to the diet.

In most cases there is no need resorting to diuretic drugs because diuretics (diuretin) are not very effective even in marked urinary disorders; many authors have completely rejected them, while others prescribe diuretin (0.1-0.2 g three times a day) and in chronic cases—thyroidin. In cases of threatening uremia limitation of liquid, temporary salt-free diet and hot packs are indicated. During convulsive seizures chloral hydrate enemas (0.5 g for very young children and 1 g for older children per enema) and spinal punctures are administered. Insulin injections (5 or 10 units) simultaneously with 5% glucose solution per os are also indicated; insulin lowers the blood pressure, stimulates excretion of sodium chloride and facilitates assimilation of sugar.

For uremia some authors recommend bloodletting (50-100 ml of blood) with subsequent subcutaneous administration of glucose solution.

A nephritis patient is permitted to walk about only after albumin has completely disappeared from the urine. In summertime the child should be kept outdoors as long as possible; warm baths are indicated after the acute stage of the disease.

After recovery the child should be long under observation, the child's urine being periodically examined.

Pyelitis

Pyelitis is a suppurative inflammation of the renal pelvis. It is most frequently observed in early childhood; up to six months of age it affects boys and girls equally; subsequently it prevails in girls. Not infrequently it is aggravated by inflammation of the urinary bladder (pyelocystitis). In some cases the renal parenchyma may also be involved (pyelonephritis).

Etiology and pathogenesis. Pyelitis is an infectious disease most frequently caused in children by the colon bacillus. In some cases infection first enters the blood and then, hematogenously, the renal pelvis; in other cases (in girls) an ascending infection is observed; the bacilli invading the renal pelvis through the external genitalia, urinary bladder and ureters. In rare cases pyelitis may be caused, in addition to the colon bacilli, by other microbes (streptococci and paratyphoid bacilli).

Pathological anatomy. The autopsy of pyelitis victims reveals distended pus-filled renal pelvis. In some cases lesion of the urinary bladder is also observed. Sometimes the interstitial tissue of the kidney is affected, which is clearly seen in microscopic section. In grave cases even the renal parenchyma is involved.

Clinical findings. Two forms of pyelitis—acute and subacute—are distinguished.

The acute form sets in impetuously and is attended with constant or remittent pyrexia. The patient's general condition is grave; older children complain of headaches. Sometimes meningeal symptoms (vomiting, convulsions, slight occipital rigidity) simulating the picture of meningitis are observed. Some cases are complicated with vomiting and frequent liquid fecal discharge. Symptoms of toxicosis resembling those of toxic dyspepsia (sunken fontanel, sunken eyes, loss of cutaneous turgor of the skin, clouded consciousness) are sometimes evident. The child is extremely pale, urinates frequently and in some cases painfully. The urine is turbid, its reaction is acid, and it contains albumin, large numbers of leukocytes which sometimes cover the whole field of vision in a microscope, few erythrocytes and motile Gram-negative colon bacilli.

Colon bacilli often appear in cultures of urine collected sterilely with the aid of a catheter.

The high temperature and grave general condition may continue for several days and sometimes even for weeks; then the temperature drops, the general condition improves and the patient begins to recover, but the pus in the urine persists in fluctuating quantities for some weeks and even months. Subsequently, pyelitis is not infrequently aggravated, especially in patients affected with influenza, pneumonia, malnutrition, etc. In cases of chronic pyelitis the affected children are underweight and pale, have no appetite and are anemic.

The subacute form of pyelitis sets in imperceptibly with normal or subfebrile temperature.

Inexplicable pallor, apathy and inappetence are the only symptoms which warrant suspicion of pyelitis in a child, a urinalysis revealing large numbers of leukocytes. The child's genitalia should be washed thoroughly before the analysis and the urine collected in a clean vessel. This form of pyelitis may also run a prolonged course and is responsible for anemia. The course of pyelonephritis is grave, a lot of albumin (more than 0.5%) and large numbers of leukocytes, erythrocytes and casts being found in the urine.

Diagnosis. Pus in the urine may also be observed in other diseases of the genitourinary tract. It may be found in patients suffering from tuberculosis of the kidney, renal calculus or renal malformations. Thorough examination for tubercle bacilli and roentgenography of the kidney (pyelography)* help to make a correct diagnosis.

The *prognosis* is relatively favourable, but sometimes acute and grave forms of septic pyelitis may end lethally, especially in younger children.

Prophylaxis and treatment. Nursing, proper feeding, good care of the child and hygiene of the girls' external genitalia (washing of the genitalia from front to back) play an important part in preventing the disease.

Treatment. During the treatment it is very important to give the child (in interims between feedings) plenty of liquid (water with fruit juices). Babies should be fed human milk (if possible) or given acidulated mixtures; they must also get enough vitamin C (fruit juices) with their food. In grave cases so-called "sugar days" are

* Pyelographic method: a roentgenographic picture is taken 10-15 minute after filling a vein with a contrast solution (sergosin); the anomalies of the kidneys, renal pelvis and ureters are clearly seen in the picture.

recommended for children past one year of age: once a week the day's diet of the child should consist of 100-200 g of sugar taken with water and fruit juice, cranberry kissel (thin jelly) or compote. The amount of liquid should be close to one litre. Daily hot baths (37-40°C) for ten minutes produce a good effect. Children should be given as much fresh air as possible (ventilation of the room, outdoor walks).

Treatment with streptocide in usual doses is recommended (p. 515). Streptocide is administered for 10-20 days until no more pus is found in the urine. Intramuscular injections with the mother's blood or measles serum in doses of 10 ml are administered every other day (a total of 5-10 injections).

Treatment with penicillin and transfusion of citrated blood of the parents or a donor are recommended in grave cases. The amount of injected blood—10 ml per 1 kg of the child's weight. In cases of renal calculus, tuberculosis of the kidney and congenital malformations surgical treatment is indicated.

DISEASES OF THE GENITALIA

Vulvovaginitis and Gonorrhea

Vulvovaginitis—a disease of the vulva and vagina—is rather frequently observed in girls, especially at an early age. This frequent affection of the vulva and vagina in these children is due to the delicate structure of these organs and their easy susceptibility to all kinds of infection.

Etiology and pathogenesis. Vulvovaginitis may be nonspecific, i.e., caused by various bacteria (staphylococcus, diplococcus, etc.) or specific, i.e., caused by gonococci.

Nonspecific vulvovaginitis is not infrequently observed in children debilitated by various infections or anemia; it is particularly often found in girls suffering from helminthiasis (oxyuriasis), when the parasites crawl from the anus (across the perineum) to the external genitalia and cause itching. In a considerable number of cases vulvovaginitis is conditioned by gonorrhea.

More often than not gonorrhea in children manifests itself in affections of the vulva and vagina, although the urethra and even the rectum are also not infrequently involved. It is most frequently observed in children between two and six years of age.

Children are usually infected with gonorrhea by their parents, relatives or persons in their immediate surroundings. In such cases the disease is contracted through sleeping in one bed with the mother who is suffering from gonorrhea, or using the same chamber-pot, basin for washing the external genitalia, sponge, towel, etc.

In exceptional cases the newborn may be infected with gonorrhea by gonorrheal mothers even during birth, but infection most frequently takes place after birth through improper washing and wiping of the babies from the infectious maternal discharges which cover their bodies and gain entrance into the vulva or eyes.

Clinical findings. Nonspecific vulvovaginitis sometimes runs an uneventful course with no general symptoms and is characterised by a mucous or mucopurulent discharge from the genitalia; an examination reveals a redness, slight swelling of the labia and vaginal entrance; a negative bacteriological test for the gonococcus confirms the diagnosis of nonspecific vulvovaginitis; vulvovaginitis disappears as the child recovers from the primary disease and general condition of the child improves.

Gonorrheal vulvovaginitis sets in rapidly and is not infrequently attended with pyrexia; the children complain of colic and pain when urinating; the vaginal discharge soon becomes purulent, sometimes greenish-coloured and corrosive to the mucosa; the mucosa of the vulva is hyperemic, the labia are inflamed and pus-covered; pressure on the lower part of the abdomen brings a considerable amount of pus from the vagina or urethra.

After affecting the vulva and vagina gonorrhea may extend to the urethra and rectum, in some cases to the bladder, ureters and kidneys, and less frequently to the mucosa of the uterus and fallopian tubes.

If no specific treatment is administered, the disease may run a protracted course (for many months and sometimes even years); as a rule, acute symptoms disappear soon, but a purulent discharge containing gonococci may persist for many months.

Diagnosis. The diagnosis of nonspecific or gonorrheal vulvovaginitis is based mainly on a detailed anamnesis and bacteriological examination; gonorrhea patients in the child's family and a prolonged course of the disease warrant the diagnosis of gonorrheal vulvovaginitis even without any examination.

Bacteriological examinations must be frequently repeated. Not only the pus which is visible to the eye, but the mucous discharge from the vagina, urethra and rectum must also be examined; this discharge may be obtained with the aid of a sterile tampon, special loop or blunt curet. As a rule, gonococci are found inside pus cells where they are located in pairs shaped like coffee beans facing one another laterally.

Prognosis. The prognosis in nonspecific, nongonorrheal vulvovaginitis is always favourable, the discharge disappearing with improvement in the child's general condition

At one time the prognosis of gonorrheal vulvovaginitis was considered rather favourable despite the prolonged course of the disease. Observations have shown, however, that inadequately treated gonorrhea in childhood is responsible for abnormal development of the genitalia, which may subsequently lead to sterility; incorrect position of the uterus and grave changes in its endometrium are not infrequently observed in women who suffered from gonorrhea in childhood.

Prophylaxis. Observance of the usual rules of hygiene and proper care of the genitalia (baths, washing) are the common prophylactic measures in nonspecific vulvovaginitis; in cases of helminthiasis the latter disease should be treated energetically. To prevent gonorrhea, it is necessary, in addition to the aforesaid, to consider the possibility of the child being infected with gonococci by the surrounding people; the child must not be allowed to share the bed, wash from one basin or use the same towel with anybody.

Children should not be admitted to children's homes, crèches, kindergartens, etc., without a preliminary medical examination, and all suspicious and sick children must be referred to corresponding institutions for treatment. All girls with acute forms of gonorrhea must be placed in special medical institutions; children who had gonorrhea before, but no longer have to be under special observation, must be placed in special children's homes or isolated from other children in view of possible relapses.

The personnel of children's institutions should be examined periodically and those discovered to have acute gonorrhea with a gonococcal discharge must be prohibited direct contact with children.

Health education of parents, teachers and the attending personnel is also one of the measures of preventing gonorrhea in children.

Treatment. Nonspecific vulvovaginitis does not require any special treatment besides general hygiene and care of the genitalia; in cases of copious and persistent discharge washing of the genitalia with 1 : 8000 solution of potassium permanganate or Burow's solution (one teaspoonful per glass of boiled water) is recommended.

Sulfa drugs, especially sulfazole, sulfathiazole and sulfadimezin are now effectively used in the treatment of gonorrheal vulvovaginitis, because they produce fewer side effects in children. Treatment with sulfa drugs is combined with penicillin therapy. A five-day course of treatment is recommended by Karysheva; penicillin and sulfa drugs for the first two days, while during the

remaining three days the child is given only sulfa drugs. Penicillin is prescribed a dose of from 400,000 to 600,000 u for the entire course of treatment; in complicated cases the dose is increased 50-100 per cent, depending on the course of the disease. The daily dose of sulfa drugs is 0.15-0.2 g per 1 kg of the child's weight and is given to the child in four or five portions.

Local therapy in the described method is of no great importance, if the foregoing method is used and consists mainly in sitz baths of camomile or weak potassium permanganate solution. In grave, complicated and protracted forms of the disease additional measures are employed, namely, gonococcus vaccination, administration of other antibiotics in combination with penicillin (streptomycin, etc.) and local application of various medicinal preparations (protargol ointment, protargol solutions, etc.) not only to the vagina, but to the urethra and rectum as well. Naturally, girls suffering from gonorrhea must be treated in hospitals and in some cases for a very long time.

In addition to the described methods of treatment, care and nutrition of the children affected with gonorrhea are of great importance; children's institutions for gonorrheal children must be of a sanatorium type so that the children may have the possibility to spend much time outdoors, engage in physical culture and sports, etc.

Phimosis. Balanitis. Hydrocele

Phimosis, i.e., elongation of the prepuce and constriction of the orifice so that the foreskin cannot be retracted to uncover the glans penis, is not frequently observed in children; in nurslings the prepuce usually adheres to the glans penis physiologically, but this adhesion does not cause any disorders and disappears by the end of the first year of life.

Only the form of phimosis in which the prepuce so tightly adheres to the glans penis that it causes urinary difficulty (dysuria) and urinary retention may be called true phimosis. True phimosis is not infrequently attended with inflammation of the glans penis and the internal surface of the prepuce so that when pressure is applied to the preputial pouch it discharges pus. Such inflammation is called *balanitis*; accumulation of the smegma and urine in the preputial pouch, which on decomposing subsequently produces purulent inflammation is an etiological factor in the development of balanitis.

Phimosis requires no treatment when it causes no disorders; only when urinary retention is observed is it necessary carefully to draw back the prepuce and eliminate the epithelial adhesions with the aid of a blunt probe; rules of asepsis must be strictly observed during this operation; the operation must be followed by applications of Burow's solution.

In *paraphimosis*—retraction and constriction of the prepuce behind the glans penis and subsequent swelling of the glans penis—the glans penis is replaced manually in a warm bath.

In *balanitis* it is necessary daily to wash the preputial pouch with Burow's solution or potassium permanganate solution with the aid of a syringe and a thin drainage tube attached to it; after that the glans penis is covered with a moist dressing.

Hydrocele is quite frequently observed in nurslings, during the first days of life; it is due to accumulation of fluid in between the connective tissue of the testis and the tunica vaginalis. On the side where fluid has accumulated, the scrotum seems enlarged and tense, and, in cases of large accumulation of fluid, it glistens and seems translucent. This tumescence of the scrotum often disappears all by itself during the first year of life and requires no surgical intervention. A surgical operation (puncture, injection of iodine solution or radical operation) is resorted to only in chronic cases, when the scrotum increasingly enlarges.

Nocturnal Enuresis

Nocturnal enuresis should be more correctly called involuntary nocturnal urination, since the urine is not discharged permanently or constantly as is the case in urinary incontinence; children urinate periodically during the night and the urine is voided in a stream as in usual voluntary urination. Nocturnal enuresis is often observed in children of preschool age; it occurs twice as often in boys as in girls.

Etiology. The etiology of nocturnal enuresis is not sufficiently clear. In most cases nocturnal enuresis is a specific manifestation of vegetative neurosis, failure to elicit a normal reflex; as a rule, it disappears before sexual maturation.

Graver forms, which do not easily respond to treatment, are observed in cases of organic lesions of the central nervous system, malformations of the spinal cord and the urinary bladder.

Clinical findings. Involuntary urination is usually observed at definite hours of the night: the urine is discharged in a stream, sometimes repeatedly in the course of the night, during sleep, and the child awakens only after urination. In grave cases involuntary urination may occur also in the daytime (diurnal enuresis).

Examination of the urine usually does not reveal any pathological deviations. Objective examinations of children show no pathological changes save some nervousness and increased excitability.

Many methods of treating nocturnal enuresis, including hypnotic suggestion have been recommended. However, there is as yet no specific single method of treating this disease. Treatment with adiurecrine has been proposed: 0.025 of adiurecrine powder has been administered into the nasal cavity once a day before sleep (A. Atabek, P. Ponomaryova). Faradisation of the urinary bladder

is sometimes affective as a psychotherapeutic method. N. Krasnogorsky recommends giving the child for the night a slice of bread with caviar, salted fish, etc., but no water; the surplus of sodium chloride is supposed to retain the liquid in the tissues and prevent accumulation of urine in the urinary bladder. A proper educational approach to the child is very important; children must not be intimidated and, especially, punished.

By constant suggestion and, in cases of older children and adolescents, autosuggestion based on elaboration of a definite conditioned reflex it may be possible to make the child awake at night and urinate.

RHEUMATISM

Rheumatism is an infectious disease associated with lesions of the heart (myocarditis, endocarditis and sometimes pericarditis) and, in some cases, with periodic acute inflammatory lesions of the joints.

FREQUENCY OF RHEUMATISM IN CHILDREN

Exact statistical data on occurrence of acute rheumatism in children are not as yet available. The frequency of its occurrence cannot be established for yet another reason, namely, the concept of true rheumatism has only lately been given scientific substantiation. Formerly the group of rheumatic diseases included joint diseases often of different etiology (this error is still common today) while heart diseases with no concomitant affection of the joints, which are, in fact, a true and most typical rheumatic infection, were ascribed to the group of heart diseases and not rheumatism.

The significant role played by rheumatic infection in children's pathology will become clear if we consider that acquired organic heart diseases in children are almost exclusively of rheumatic etiology and that chorea minor is also one of the typical manifestations of rheumatism.

According to various authors, acquired heart disease of rheumatic etiology occurs in children of school age on an average in 1-2 per cent of all cases. Rheumatism is more widespread among older children (8-15 years of age), while in early childhood the rate of rheumatic morbidity is much lower.

There is no special difference between boys and girls as regards the frequency of rheumatic diseases; the incidence of chorea minor is higher in girls.

According to some data, the morbidity rate is four times as high in families affected with rheumatism as it is in nonrheumatic families.

Etiology and pathogenesis. The pathogenic agent has not yet been established. Many authors ascribe the role of pathogenic agent in rheumatism to one of the streptococci. As a rule, streptococci are found in the blood and the heart valves of affected persons only in cases of *endocarditis lenta* which is a septic phase of the rheumatic process.

However, the specific role of streptococci has not yet been demonstrated and no special variety of "rheumatic streptococcus" has been isolated. The disease is supposed to be caused by a virus.

Some authors suggest another theory of the etiology of rheumatism. They consider rheumatism a result of an allergic reaction* of the tissues and the whole organism, sensibilised by some nonspecific substances of an albuminous nature, which gained entrance into the organism from the bacteria before.

In the opinion of some authors, the presence of a specific microbe is not necessary for diagnosing acute rheumatism, inasmuch as various microbes and various proteins may evoke in a patient a reaction which will condition a characteristic rheumatic affection. Many authors, however, do not agree with this concept.

As for the special allergic state of the organism in acute rheumatism, which produces a sufficiently specific and characteristic picture of the disease, this reaction is apparently induced by a special and characteristic, but as yet unknown, infectious factor which possesses allergenic properties, as is also the case in many other specific infections which cause at a certain stage of their development a special allergic condition characteristic only to the given infection (scarlet fever, tuberculosis). It may therefore be quite justifiably assumed that rheumatism is an infectious entity, the streptococcus playing but an accessory role.

Pathologic anatomy. Since 1904, when Aschoff, a German pathologist, discovered special nodules—granulomas—arranged along the course of the vessels in the heart, and Talalayev, a Russian pathoanatomist, made a detailed study of them, the essence of pathoanatomical changes in true rheumatism has been fully established.

These granulomas appear as a result of the action of a specific virus on tissue (heart tissue in particular). Granulomas are an accumulation of large cellular elements surrounded by a zone of lymphocytes.

The development of connective tissue along with destruction of muscular tissue elements causes the appearance of these granulomas.

These specific granulomas are not found in affected joints, where edema, which is considered to be a peculiar reaction of the organism with no specific lesion of the joint, is mostly observed.

The so-called nonspecific allergic inflammation of the heart muscle which usually conditions the transition of acute processes to decompensation is very characteristic of rheumatism.

Changes in the cells of the corpus striatum are found in chorea which is practically a rheumatic encephalitis.

* An allergic reaction is a state of hypersensitiveness of the organism to certain substances, mainly of an albuminous nature, the organism reacting more intensely, especially if repeated ingestion has made the organism increasingly sensitive to them, i.e., has sensibilised it.

CLINICAL FINDINGS

The symptoms of acute rheumatism are very variable. In typical cases the disease begins suddenly, articular phenomena (pain, tumescence) sometimes appearing first.

General symptoms—inappetence, not infrequently high temperature—are observed from the very first days, while articular phenomena are sometimes so negligible and transient that they may be overlooked. In some cases children complain only of pain in the joints but no pathologic changes are observed in them. Not infrequently the joints are hot to touch.

The knee, ankle, shoulder and small finger and toe joints are mostly affected. Pain and tumescence often appear now in one and now in another joint (the form of the disease associated with polyarthritis). The spine, especially its cervical vertebrae, may also be involved.

Various eruptions (hemorrhages, erythema papulatum) are sometimes observed on the skin.

The heart is so frequently involved (in 80-90 per cent of the cases) that this is considered a most characteristic feature of rheumatic infection. True, heart affections are sometimes insidious and may be overlooked in the beginning of the disease, but in 75 per cent of the cases the symptoms of heart affection are sufficiently clear and the clinical picture leaves no doubts of developing heart disease (rheumatism of the heart).

In some cases, however, there are “pure” forms (about 20 per cent) when only the joints are affected.

Rheumatic infection affects all three layers of the heart (endocardium, myocardium and pericardium); *clinically* it may manifest itself simultaneously in endocarditis, myocarditis and pericarditis. In most cases, however, only one layer, more frequently the myocardium or endocardium, is clearly affected.

Endocarditis. Inflammation of the endocardium, being a most characteristic feature of rheumatic infection, is, in its anatomical essence, a peculiar process in the valves and the parietal endocardium, leading to development of specific granulomas, disorders of the valvular functions and subsequently to their shrivelling.

In most cases fibrinous endocarditis results in changes in the valves leading to stable heart diseases, which may involve the different heart valves. The valves most frequently affected in rheumatism may be arranged in the following order: bicuspid valve, aortic valve and tricuspid valve.

Lesions of the pulmonary valve are exceptionally rare. In some cases there are combined lesions as well. The most usual combination is mitral-aortic; simultaneous affection of all the valves (exclusive of those of the pulmonary artery) occurs less frequently.

According to modern views, rheumatism affects simultaneously both the endocardium and myocardium (endomyocarditis), which may lead, especially in children, to circulatory disorders.

In young children who are affected with rheumatic endocarditis extremely rarely, the onset and course of the disease are of a septic character; the disease is very acute with no immediate local manifestations in the heart (murmurs, dilatation of the heart). Increasing general pallor, temporary attacks of cyanosis, rapid respiration, small and quick pulse are observed as a rule; the child usually dies with symptoms of cardiac weakness, the autopsy sometimes revealing endocarditis.

In older children the onset of rheumatic endocarditis is marked by weakening of the first sound at the apex which subsequently changes into a systolic murmur with an accented second sound at the pulmonary artery and dilatation of the heart. Subjective sensations—pain and spasm in the region of the heart are at first usually very mild, or even absent. Stable tachycardia is a constant symptom of endomyocarditis.

A malignant and rapid course of endocarditis is marked by chills, very early appearance of murmurs and dilation of the heart.

In children endocarditis runs a protracted and progressive course; according to modern anatomical findings, the acute symptoms do not subside in less than 3-5 months, but the murmur and dilatation of the heart become permanent. Only in rare cases the disease is limited to one attack; usually the process continuously progresses and is frequently aggravated, thus leading to invalidity.

If the disease does not end lethally, it soon develops into a permanent heart disease with possible recurrences of the rheumatic infection and new aggravation of the process in the heart.

Myocarditis. Inflammation of the myocardium is almost always associated with rheumatic affection of the heart.

Anatomically rheumatic myocarditis is classified with the group of so-called interstitial myocarditis; it is characterised by peculiar cellular nodules (rheumatic granuloma) in intermuscular connective tissue. This process results in myocardial sclerosis with possible subsequent change in the whole, or part of the muscular and conduction system of the heart. Rheumatic myocarditis, like any

other forms of the disease, is characterised in its more pronounced form by the same *clinical symptoms*: pallor, dyspnea, sometimes cyanosis, small accelerated pulse, weakened heartbeat; enlargement of the liver is also not infrequently observed.

Inasmuch as myocarditis is rarely the only concomitant of rheumatic infection, the symptoms of myocarditis usually merge with those of endocarditis into one clinical picture of heart disease.

Pericarditis. Pancarditis. Pericarditis is a frequent phenomenon in rheumatic infection, particularly if it is associated with myocarditis and endocarditis, thus producing a clinical picture of pancarditis (general inflammation of the heart).

Anatomically rheumatic pericarditis is characterised by the formation of a serofibrinous (sometimes purulent) exudate in the pericardium, as well as the epicardium.

Eventually, formation of an exudate may take place which results in pericardial adhesion and obliteration.

Pericarditis which complicates symptoms of myocarditis and endocarditis in rheumatic infection is at first characterised by the appearance of friction murmur of the pericardium; in cases of exudation, a gradual increase in cardiac dullness and sometimes bulging of the precordia are observed.

Exudation in rheumatic pericarditis is less than in tuberculous pericarditis in which the heart bulges out more perceptibly and more often.

In some cases of rheumatic infection (3-4 per cent) rheumatic nodules located in subcutaneous tissue are observed; these nodules most frequently occur in the regions of joints or on galea aponeurotica. They are associated with rheumatism, whose course is characterised by clearly marked general symptoms (malnutrition, anemia, and fever).

The form of rheumatism with concomitant formation of nodules is mostly grave and its prognosis is more unfavourable. Rheumatic infection, whatever its form, is not limited to one acute attack; after a remission the attacks recur and again *alternate with remissions*. Acute attacks follow each other aggravating the child's condition and often lead to death.

The clinical picture of remissions is not very clear as yet and, as a rule, no other affections save compensated heart disease and anemia are observed. Children with these symptoms are considered healthy, but the rheumatic process may continue and some time later provoke a new attack of the disease. It is only during attacks that a characteristic clinical picture of an acute form of the disease is observed. In virtue of this fact it would be more correct

to call-rheumatism, like tuberculosis, syphilis and malaria, a chronic infection.

Chorea. In addition to the aforementioned clinical manifestations of rheumatism children are affected with one more form of the disease which for a long time has not been regarded as a rheumatic infection. This form is *chorea minor*.

The disease is characterised by involuntary and irregular action of the muscles with peculiar disturbances in neuromuscular coordination. Affected children are constantly moving: now they shrug their shoulders, now strangely twist their arms and then quickly bend their heads; in some cases the facial expression continuously changes, the face alternately assuming a laughing, gay, confused, worried, frightened or cunning expressions.

In grave cases of chorea children can neither sit nor stand; they cannot dress or eat by themselves and often have speech disturbances. All these involuntary movements usually cease during sleep. The disease is connected with an affection of a certain part of the brain and often occurs together with affection of joints and the heart; as a true rheumatic infection it produces not one but several attacks. The attacks occur over a period of several months (one and a half to four months), sometimes without affections of the heart, terminate rather favourably; in other cases, i.e., combined affection of the heart and joints, the picture of the disease is very grave and the prognosis is very poor. As was already mentioned, chorea affects girls more frequently than boys.

Diagnosis. It is not difficult to diagnose acute rheumatism which is so characteristically manifested in affections of the heart, considering that no other children's infection could cause such permanent cardiac changes. Almost every children's heart disease (not congenital) is one of the links of rheumatic infection. Only grave septic affections which usually end lethally may produce a picture of septic endocarditis, while persistent heart diseases are a result of rheumatic infection suffered in childhood.

As for the affections of joints, children sometimes also have chronic arthritis of nonrheumatic origin. This form of arthritis clearly differs from rheumatic arthritis which usually passes rapidly and produces no organic changes in the joints, while chronic arthritis of nonrheumatic origin usually runs a protracted course and deforms the joints; in some cases the joints become ankylosed, which is never observed in rheumatic arthritis.

The diagnosis of rheumatism in cases involving formation of the aforementioned nodules is incontestable since nothing resembling these nodules is observed in any other disease.

Nor is it difficult to diagnose chorea because of the characteristic involuntary muscular movements, especially if the disease includes affections of the joints and heart.

Prognosis. Stable changes in the heart and the possibility of repeated attacks warrant a very serious prognosis.

The prognosis in rheumatism aggravated with pericarditis in a patient suffering from heart disease is very unfavourable; pancarditis often causes death, but sometimes the patient's condition may improve (the process subsides and cardiac compensation is restored).

Rheumatic infection is one of the principal causes of death among older children.

Rheumatism causes death much less frequently in early childhood, although the younger the child the higher the mortality.

TREATMENT AND CONTROL OF RHEUMATISM IN CHILDREN

Strict confinement to bed for a comparatively long time is of primary importance in treating rheumatism.

Short-term treatment in a hospital is not enough for the process to subside fully considering that an acute attack of rheumatism is not infrequently attended with a specific inflammatory process in the heart, which lasts 2-3 months. Rheumatic children require longer treatment with a regimen approximating to that of a sanatorium which is the only way to obtain cardiac improvement and reduce the number of relapses.

The child may not be allowed out of bed before one and a half to two months have elapsed since the onset of the disease, i.e., the period required for the inflammatory processes in the heart to terminate naturally.

Of medicinal preparations sodium salicylate is most widely used in the treatment of rheumatism; some authors consider it a specific antirheumatic drug.

Sodium salicylate is prescribed in a dose of 0.5 for every year of the child's age, the daily dose being divided into 5-6 portions (the dose for a 10-year-old child is 1 g 5 times a day). Sodium salicylate treatment is administered for rather a long time—one to one and a half months; the full dose is administered during the acute stage of the disease, but as the clinical symptoms begin to subside the patient is given half the dose for a period of two weeks. It should be noted, however, that, as practice has shown, sodium salicylate does not prevent development of the pathological process in the

heart, the preparation acting mainly as an analgesic in joint affections. The side effects often produced in children by sodium salicylate are epigastric pain, nausea, vomiting and diarrhea. Children are, therefore, more often prescribed pyramidon in a daily dose of 0.15-0.2 g for every year of age (but not exceeding 2 g) to be taken in three or four portions a day for a period of six weeks; when the symptoms of acute processes have abated the dose of pyramidon is cut in half.

The entire course of treatment takes two months. The appearance of red urine does not contraindicate the treatment and is merely a result of chemical colouring by pyramidon.

Prophylaxis usually consists in a general improvement of the living conditions, a proper regimen for rheumatic children during the remissions and oral hygiene. Some authors recommend a tonsillectomy in cases of frequent tonsillitis.

An earliest possible diagnosis of cardiac changes and treatment until the inflammatory rheumatic process has completely subsided are necessary to prevent frequent recurrence of rheumatic attacks.

After discharge from the hospital the children should be kept under systematic observation; in cases of aggravation the hospital treatment must be repeated.

State and public control of rheumatism. All measures aimed at controlling rheumatism should be carried out on a country-wide scale.

A Committee for Studying and Controlling Rheumatism and Joint Diseases has been organised in the Soviet Union, under the U.S.S.R. Ministry of Public Health; some of the Union Republics have republican, territorial and regional committees.

Special rheumatic departments and wards have been set up in many children's polyclinics, hospitals and sanatoriums. Polyclinical departments of children's hospitals have special antirheumatic offices which must keep records of all rheumatic children who live in the district served by the department, keep them under constant observation after their discharge from hospitals, help to organise their home regimen and watch them at school.

Special attention should be devoted to observing a proper regimen of rheumatic children at school. School physicians must keep a record of all such children as well as those suspected of having the disease; together with the physicians from district polyclinics they must conduct regular check-ups and examinations of such children.

Children frequently affected with tonsillitis, as well as those who recently had scarlet fever, should be included because, as

observations show, rheumatism affects such children more frequently.

A usual school regimen is too difficult for a child who has just returned to school after his discharge from a hospital. In such cases the child should have an easier regimen for a certain period of time, i.e., additional days off, etc.

Rheumatic children must not be exempt from physical culture activities, but must be given exercises corresponding to their physical abilities. Physical exercise and properly chosen sports are as necessary for rheumatic as for healthy children.

TUBERCULOSIS

At the end of last century tuberculosis in children was considered a rare disease. All studies in tuberculosis conducted at that time were devoted mainly to its manifestations in adults; affected children were mentioned merely in passing. Subsequently, it was found that tuberculosis begins in childhood.

It is therefore clear that the study of tuberculosis in children is of decisive importance for regular prophylaxis and control of the disease. Incipient tuberculosis in children has certain features which differ from those of the terminal stage of tuberculosis in adults. Tuberculous infection in adults is very widespread: post-mortem examinations of people who died of various diseases sometimes reveal old and emperceptible tuberculous lesions.

Incidence of tuberculosis in children. Autopsies of children who died of various diseases not infrequently reveal tuberculous lesions.

Since 1907, when Pirquet suggested a simple method of intravital detection of tuberculous infection by means of a skin reaction to inoculation with tuberculin, it has become possible to estimate the incidence of tuberculosis in children.

Etiology, ways of infection and pathogenesis. In 1882 Robert Koch discovered a special bacillus which is the pathogenic agent of tuberculosis.

Tuberculosis is an infectious disease and people are infected mainly from each other.

The tubercle bacillus (*Mycobacterium tuberculosis* var. *hominis*) is essentially the most frequent causative agent of tuberculosis. However, it is possible to infect children with the tubercle bacillus of cattle (*Mycobacterium tuberculosis* var. *bovis*), the infection gaining entrance into the child's body through the alimentary canal with the milk.

Autopsies show that 1 per cent of children are infected with the latter variety. Some authors consider the percentage to be even higher.

Infection with the *Mycobacterium tuberculosis* var. *homin*i occurs mainly by the air-borne method, i.e., as a result of tubercle bacilli entering the smallest ramifications of the bronchial tree through the respiratory tract.

Patients with an open form of tuberculosis are the most frequent source of infection. In this case people become infected either through droplets of sputum discharged into the air during talking and, particularly, coughing (droplet infection), or through particles of desiccated sputum coming from contaminated things (handkerchiefs, linen, clothing, upholstered furniture, etc.).

The possibility of infecting children with tuberculosis by adults increases if adult patients maintain close contact with them (a common bed, kissing, eating from the same dishes, etc.). The question of an infant being infected in utero is not disputed. In cases of intrauterine infection the picture of the disease develops as follows: the child develops a primary tuberculous lesion (usually pulmonary) in the form of a so-called primary tuberculous focus (primary focus) which heals in the overwhelming majority of cases (autopsies reveal scars characteristic of the primary tuberculous focus in 90 per cent of adults); the lymph nodes in the root of the lung are affected simultaneously. In some instances this healing process does not take place and subsequent generalisation (dissemination to other organs) through lymphatic or circulatory system occurs; this dissemination may be general and result in miliary tuberculosis. Sometimes only separate organs (lungs, bones, skin, etc.) are involved. Such is the course of tuberculosis in children.

Adults may have secondary infection (reinfection), in which case the tuberculous process becomes chronic, mainly in the upper lobes of the lung (pulmonary tuberculosis). Only older children or adolescents may have chronic tuberculosis.

Immunity. The organism of the child affected with tuberculosis carries this infection all through life, and yet adequately resists it.

Low living standards and such diseases as measles, whooping cough, influenza and pneumonia constantly threaten to activate the tuberculous foci. Under normal living conditions, these inactive tuberculous foci, surrounded by a fibrous capsule and calcified, guarantee the patient against the constant threat of reinfection from the infected environment.

If tuberculosis appears in localities for the first time, it spreads rapidly among the entire population, affecting both children and adults, and runs a course of an acute (rather than chronic) infection, like typhus, cholera, etc. This is due to the fact that the people who had no contact with tuberculous infection in childhood have not acquired immunity against tuberculosis.

PRIMARY COMPLEX

The primary complex is an affection of the lung in the form of a primary tuberculous focus (primary focus), which in turn affects regional lymph nodes. The primary focus is an exudative inflammatory focus in pulmonary tissue.

Such tuberculous foci in the lungs are usually single and only in 4-5 per cent of the cases—multiple. The primary affection of the lungs is usually resorbed, leaving a small scar which microscopic examination sometimes reveals at autopsies. Eventually, this primary focus becomes calcified (the so-called Ghon lesion).

The primary complex often develops imperceptibly which makes it difficult to establish its development clinically. Only a positive Pirquet reaction in an infant warrants its assumption. In some cases caseous tissue disintegration takes place at the site of the primary complex in the lung and even a cavern may be formed (in infants up to one year of age).

In cases of a positive Pirquet reaction anamnestic data on the nursing's contact with tuberculous patients (parents, relatives, persons in immediate surroundings) confirm the existence of the primary complex.

TUBERCULOUS BRONCHADENITIS

As was already mentioned, tuberculous infection is disseminated from the primary focus through the lymphatic vessels to the adjacent lymph nodes of the root of the lung and causes tuberculous bronchadenitis. Bronchadenitis may vary in its clinical manifestations with the intensity of the tuberculous process in the lymph nodes and the size of the lesion. It may be attended with a perifocal inflammation. This type of bronchadenitis is called infiltrative.

The affection of the lymph nodes may be tumorous when something like a tumour is formed in a lymph node which becomes considerably enlarged. This type of bronchadenitis is known as *tumorous* and its clinical manifestations differ from those of infiltrative bronchadenitis.

The general condition of the child affected with the aforementioned types of bronchadenitis deteriorates considerably; the symptoms include emaciation, inappetence, failure to gain weight or loss of weight, general sluggishness and pyrexia.

All these symptoms are not so clearly marked and not so persistent in infiltrative bronchadenitis, as they are in the tumorous

form of the disease. The temperature in some cases of tumorous bronchadenitis averages up to 38-39° for a long time. This type of bronchadenitis occurs more frequently in early childhood. In addition to the afore-described general symptoms, infants affected with bronchadenitis develop a characteristic cough resembling whooping cough, two sounds being heard during every cough: a coarse, low-pitched sound and a high-pitched sound (bitonal cough).

Roentgenological examination of the affected child reveals enlarged lymph nodes in the form of oval shadows. Anatomically, the affected lymph node in tumorous bronchadenitis is markedly enlarged, with a caseous inflammation inside and an infiltrate surrounding it.

In diagnosing the disease it is important to remember that the lymph nodes may also be enlarged in other infectious diseases and that in some cases they may be enlarged for a rather long time. These diseases include influenza, measles, and sometimes whooping cough; a thorough anamnesis showing that the child had contact with tuberculous patients and positive tuberculin tests (Pirquet and Mantoux tests) make it possible to determine the nature of bronchadenitis.

Chronic tuberculous intoxication may also be considered a form of tuberculous bronchadenitis which has no clearly defined local lesions.

Prognosis. The prognosis in tuberculous bronchadenitis is mostly favourable: in tumorous bronchadenitis the caseous masses are usually reabsorbed and a scar is formed on the site of the inflammation, the tuberculous foci in the lymph nodes not infrequently growing calcified.

In infiltrative bronchadenitis, after abatement of the perifocal inflammation, the lymph nodes become cicatrised and calcified, which can be demonstrated roentgenologically. In rare cases tumorous bronchadenitis may result in disintegration of the lymph nodes with subsequent development of tuberculous pneumonia.

CHRONIC TUBERCULOUS INTOXICATION

In some cases children already infected with tuberculosis show no symptoms of the disease for a long time; only a positive Pirquet test is indicative of the tuberculous infection. At first the affected child in no way differs from healthy children, but later the first signs of the disease, manifested in general disorders, ap-

pear; the child begins to lose weight and appetite, easily tires and at times runs a fever for no apparent reason; sometimes the child coughs.

Objectively, no pathological changes in the lungs and other organs are observed, or the deviations from normal are slight. In these children the Pirquet reaction is positive and a slight enlargement of the lymph nodes is sometimes discovered roentgenologically. This form of tuberculosis with no clear localisation is called chronic tuberculous intoxication; it was first described by Kisel who gave a detailed description of its symptomatology.

Sometimes this condition is not very clearly defined and the child lives the life of a healthy child; this is the first stage of chronic tuberculous intoxication—I₁.

If these disorders are strongly pronounced, the child fails to gain weight, lags in physical development, has stable malaise and headaches and is permanently indisposed; it is the second stage of chronic tuberculous intoxication—I₂.

This group of chronic tuberculous intoxication I₁ and I₂ leads all other tuberculous affections in children (up to 94 per cent), while other clearly localised forms occur in 4-6 per cent of all cases. This disease is usually attended with changes (enlargement) in the lymph nodes of the root of the lung.

Peripheral lymph nodes also change in cases of chronic tuberculous intoxication; they become solid and are sometimes palpated in places where they are not usually observed in healthy children (*thoracic* on the anterior surface of the thorax, *subclavicular*, *ulnar*).

Sometimes these nodes are palpated very early, even in nurslings; other infections, such as syphilis, skin diseases, etc., being excluded, this is usually indicative of tuberculous infection.

Thus, the localisation of tuberculosis is essentially indefinite and phenomena of a general character usually come to the fore. Chronic tuberculous intoxication is not a result of some stable process; it may either fade gradually, the child seeming practically healthy and only the Pirquet test revealing the presence of tuberculous infection in the organism, or the process will subsequently become clearly localised in some organ and one of the forms of localised tuberculosis described below will then develop.

In nurslings chronic tuberculous intoxication is of a somewhat different character, but essentially it differs very little from that in older children. Usually, during the first months of life no difference is observed between babies born of a tuberculous mother and those of healthy mothers; the newborn may be of normal weight

(however, the weight of some of these babies is below normal) and for the first 3-4 months may develop normally; in some instances small consolidated lymph nodes are palpated as early as during the first days of life. Eventually the baby begins to lag behind in weight; its mother complains that the baby "catches cold" all the time, coughs frequently and often suffers from influenza; inappetence and feebly marked anemia develop. By this time the Pirquet reaction becomes positive and the picture of a tuberculous affection, sometimes with no definite localisation (chronic tuberculous intoxication) is on hand. In other cases general miliary tuberculosis develops very rapidly. The clinical manifestations of the primary period of pulmonary affection in children are barely noticeable; usually it is the secondary manifestations of tuberculosis that have to be considered when the infection extends from the primary focus to other organs, including the lungs. The following description of localised tuberculosis in children therefore embraces all forms of secondary dissemination of the process through various organs, which is mostly observed in children of approximately up to ten years of age. The forms of chronic pulmonary tuberculosis (consumption) in older children and adolescents do not essentially differ from those in adults.

INFILTRATIVE PULMONARY TUBERCULOSIS

Two types of tuberculosis affection are now implied by this term; pulmonary infiltrations and pulmonary infiltrates.

Pulmonary and hilus-pulmonary infiltrations are perifocal inflammations around a primary tuberculous focus in the lungs or around a lymph node of the root of the lung resembling a reactive inflammation.

This type of inflammation surrounding tuberculous foci has no typical histological structure of a tuberculous lesion (tubercles, giant cells), but etiologically is undoubtedly a process associated with tuberculosis.

The onset of the disease is marked by pyrexia; percussion of the lungs reveals intense dullness in the interscapular space, sometimes involving a whole lobe of the lung, while auscultation reveals weakened respiration with perhaps a few moist rales only on the periphery of the dull region. Despite the extensive affection of the lungs no dyspnea is usually observed.

In cases of small infiltrations percussion and auscultation fail to detect any local phenomena in the lungs and the nature of the disease can be established only by X-ray examination.

The peculiar feature of pulmonary infiltrations also confirmed by X-ray examinations is that they are usually completely re-sorbed, even if after a rather long period of time (several months), leaving connective tissue strands. Only in rare cases are caseosis or fibrosis observed in the lungs.

A *pulmonary infiltrate* is a perifocal inflammation developing as a reinfection about a new tuberculous focus or around an old activated focus. Its characteristic feature is a tendency to rapid decomposition during which the nature of the disease is usually established. Until then the onset of the disease is marked by pyrexia, faster erythrocyte sedimentation rate and changes in the hemogram (to the left); neither changes in the lungs can be detected either by auscultation or percussion. Infiltrates are more frequently observed in older children and adolescents while infiltrations affect mostly infants.

ACUTE CHEESY PNEUMONIA

Acute cheesy pneumonia develops as a result of a *rupture of a large infiltrate* or softened gland into a big bronchus or the blood stream. The clinical symptoms are the same as in croupous pneumonia: rapid onset, pyrexia, obtusion in some part of the lung, cough with sputum containing tubercle bacilli. The course is rapid, death occurring in the middle or end of the second week.

CHRONIC CHEESY PNEUMONIA

Chronic cheesy pneumonia develops gradually, the temperature long remaining normal and a slight cough sometimes observed.

The process usually originates in caseously degenerated bronchial glands whence the affection extends to the adjacent pulmonary tissue.

Examination of the lungs usually reveals obtusion, more often in the region of the root of the lung, but sometimes also in the lower lobe. Respiration is, as a rule, weakened and no rales are auscultated, for which reasons tuberculosis is often mistaken for exudative pleurisy.

Sometimes the process drags on for months, causing gradual emaciation and anemia. At the terminal stage of the disease signs of softening of the cheesy masses (moist rales, cavern symptoms) appear and the children die of progressive emaciation.

The disseminated form of pulmonary tuberculosis caused by multiple formation of small and medium-sized foci corresponds in its clinical manifestations to chronic bronchitis or focal pneumonia. It is difficult to differentiate this type of pulmonary tuberculosis from nonspecific focal pneumonia. The anamnesis, Pirquet test, roentgenoscopy and examination of the sputum help to diagnose the disease.

This type of tuberculosis is now successfully cured by early treatment with streptomycin.

The cheesy-cavernous form of pulmonary affection as a result of tissue decomposition also occurs in children, the small caverns producing no characteristic symptoms (tympanitis, amphoric respiration). Caverns the size of a pea can be revealed only by X-ray examinations.

The prognosis in this form of pulmonary affection in children is usually poor, although cases ending in recovery are also sometimes observed.

Pulmonary hemoptysis is very rare in children. In cases of hemorrhages from the throat it should be remembered that children not infrequently bleed from the nose, the blood being discharged mostly through the mouth.

TUBERCULOSIS OF THE PLEURA

Pleural lesions are often associated with pulmonary tuberculosis: *fibrinous layers on the pleura* and pleural adhesions are found almost in all protracted cases of chronic pulmonary tuberculosis.

Dry pleurisy is not diagnosed very often. Sometimes in a generalised process an autopsy may reveal tubercles on the pleura. In other cases *exudative pleurisy* is observed, the exudate being serous (more often) or serofibrinous.

It should be noted that the majority of serous pleurisies are of tuberculous etiology. That is why in practice every case of serous pleurisy, which sometimes appears suddenly, should be regarded as a sign of an active tuberculous process in the child. A thorough examination of the exudate not infrequently reveals tubercle bacilli. and in many cases it is possible to diagnose tuberculosis by way of inoculating guinea pigs.

Formation of serous exudate is considered a serious signal attesting a severe outbreak resulting from a hematogenic infection from some tuberculous focus in the organism.

In some cases serous pleurisy is characterised by gradual onset of the disease; it is marked by fatigue, inappetence, fever, pain in

the side; usually, however, the onset of the disease is acute, the temperature of the child until then apparently quite healthy rising to 40°. In cases of sufficient accumulation of exudate, obtusion and weakened respiration in the affected side are observed, while in cases of large accumulations of exudate the adjacent organs may be displaced. In doubtful cases, pleural puncture is of decisive importance.

The course of serous pleurisy is mostly favourable; the disease lasts 3-6 weeks and results in recovery, but subsequently children who had serous pleurisy may show symptoms of a developing tuberculous infection.

TUBERCULOSIS OF THE LARYNX

Tuberculosis of the larynx is found in children extremely rarely; it occurs as a secondary affection attending a protracted course of pulmonary tuberculosis, caseous tuberculosis in particular.

Symptoms of laryngeal lesion: pain on swallowing, hoarseness, tormenting cough, diffuse redness and tumescences around one or several small ulcers or around several tubercles which may be revealed by special examination of the larynx.

The prognosis depends upon the main lesion of the lungs.

TUBERCULOSIS OF THE PERIPHERAL LYMPH NODES

The changes in the lymph nodes in tuberculous infection have already been discussed; the disease is not properly tuberculosis of the glands, but is one of the symptoms of tuberculous infection. In some cases, however, the affection of the lymph nodes of a tuberculous character may be considerable, thus constituting a localised process. Most often it is the cervical lymph nodes that are affected, but affection of the inguinal and axillary glands is also sometimes observed. The nodes become considerably swollen, sometimes reaching the size of an egg, and are painful to touch. At first they are dense, but later soften and suppurate. The pus effuses to the exterior and forms fistulas which persist for a long time; in the end the fistulas heal and form characteristic irregular scars. Sometimes the process may drag on for months and in older children even for years.

In other cases effusion does not occur and the inflamed nodes undergo fibrous degeneration, the glands adhere to each other

and their surface becomes tuberosus due to development of cicatricial tissue.

In the majority of cases the outcome of tuberculous adenitis is favourable.

TUBERCULOSIS OF THE INTESTINES AND PERITONEUM

Tuberculosis of the intestines is observed either in secondary infection by the sputum containing tubercle bacilli (pulmonary tuberculosis) or in generalised tuberculosis (hematogenic infection). Primary infection of the intestines is sometimes also observed as a result of consumption of food contaminated with tubercle bacilli (milk of tuberculous cows, etc.).

The pathoanatomical changes in the intestines consist in formation of tuberculous ulcers mostly in the lower part of the small intestine, in the region of the Peyer's patches.

Tubercles develop under the epithelium and subsequently merge, undergo caseous degeneration and disintegrate, forming ulcers with serrated margins. The ulcers may spread as far as the serous membrane, which becomes inflamed and tuberculated; subsequently they adhere to the adjacent intestinal loops. In favourable cases the ulcers may cicatrise and form strands and strictures.

At an early age the disease may run a symptom-free course, as atrophy without diarrhea. In some cases there is diarrhea, sometimes sanguineous; not infrequently it alternates with constipation. General nutrition is sharply disturbed, and the infants often die of emaciation.

The mesenteric lymph nodes may also be involved in the process if there are tuberculous lesions in the adjacent peritoneal cavity (tuberculosis of the intestines or peritoneum). Sometimes, however, an independent form of tuberculosis of the mesenteric lymph nodes is observed, an autopsy revealing the latter in the shape of huge pouches (mesenteric lymphadenitis).

A caseously degenerated lymph node may soften and form an abscess which ruptures into the peritoneal cavity or intestines. The affected glands may sometimes be revealed by palpation of the abdomen.

In cases of suppuration there are sharp pains as in appendicitis. If the glands are but slightly enlarged and do not suppurate, no symptoms, except general changes characteristic of tuberculosis and a positive Pirquet reaction, are evident.

Tuberculous peritonitis (inflammation of the peritoneum) is observed either as a process extended from the intestinal wall and

mesenteric glands or as an independent tuberculous affection of the peritoneum in general tuberculous infection.

It may be *exudative*, like pleurisy, in which case a large amount of serum accumulates in the abdominal cavity and the abdomen becomes considerably distended. The disease runs a comparatively favourable course and ends in recovery.

The second form of peritonitis—*adhesive*—is marked by inflammation on the peritoneal surface which results in adhesions of the intestines, causing difficulty in digestion and leading to emaciation. Clinically, unevennesses may sometimes be found on the surface of the abdomen; in some cases a kind of swelling, in the form of a strand running across the abdomen (thickened and shortened omentum) may be palpated.

This form of the disease often ends lethally.

There is one more, the third form of the disease—*ulcerative peritonitis*—marked by formation of ulcers and fecal fistulas. This form of peritonitis almost always results in death.

TUBERCULOSIS OF THE KIDNEYS, LIVER, AND SPLEEN

Tuberculosis of the kidneys in children is a rare disease compared with its incidence in adults. A tuberculous lesion of the kidney is characterised by the appearance of pus in the urine, pyuria ordinarily persisting for a very long time; usual urinalysis often fails to reveal any microbes, tubercle bacilli being discovered by a more careful analysis; sometimes the tuberculous character of the affection may be established by inoculating animals (guinea pigs) with the urine of affected children. A positive Pirquet reaction and, in some cases, palpation of the enlarged kidney and hematuria (blood in the urine) help to diagnose the disease. A final diagnosis is made by urological examinations (cystoscopy, pyelography, roentgenoscopy of the kidney with sergosin [skiodan]).

The *prognosis* in tuberculosis of the kidney is usually grave. Some forms of the disease produce no symptoms and end in recovery; autopsies of consumptives reveal cicatrices in the kidneys as a consequence of healed foci of secondary tuberculosis (A. Kisel). Administration of streptomycin, phthivazide (isonicotinic acid hydrazide derivative) and other antituberculous preparations make it possible to cure tuberculosis of the kidney.

Tuberculous lesions of the liver and spleen are not separate disease entities; formation of tubercles on these organs is a fre-

quent phenomenon associated with general miliary tuberculosis; except some enlargement of these organs, the tuberculous lesion of the liver and spleen produces no clinical symptoms.

TUBERCULOSIS OF BONES AND JOINTS

Tuberculosis of the bones and joints is a frequent affection of childhood, developing as a secondary disease caused by penetration of tubercle bacilli from the primary focus with the blood.

Various bones and joints may be affected by this disease. Patho-anatomically the affection is marked by the appearance of tubercles in the bone, at sites of arterial ramifications (near the epiphyses); subsequently the bony tissue undergoes caseation, and caverns in the bones and sequestra develop. The periosteum is also involved, and the peripheral parts of the bone sclerose. Pus is discharged from the tuberculous foci, cold abscesses and external fistulas appear and persist for a long time. Lesions of the joints gravely affect the functions of the extremities, and ankylosis develops.

Tuberculosis of the bones and joints is classified among surgical diseases; all forms of this disease and the methods of its treatment are described in detail in textbooks of surgery.

The severest form of bone affection is tuberculosis of the vertebrae (tuberculous spondylitis). In case of late diagnosis and absence of treatment it results in considerable and stable deformation of the thorax.

The onset of tuberculous spondylitis is marked by pain which appears in some part of the spine; then the spinous processes of the vertebrae bulge out and a hump develops. Local destruction of the bone and pyorrhea take place; wandering abscesses on the back, thigh or neck are formed.

The shape of the tuberculous humpback differs from that of the rachitic curvature of the spine; in rickets the curvature is arciform, while in tuberculous spondylitis it is angular. If the child is placed in a prone position and is lifted by its legs the rachitic hump straightens out, while the tuberculous hump does not. Moreover, in far advanced cases of tuberculosis of the spine the destroyed vertebrae not infrequently compress the spinal cord, producing a picture of a spinal cord affection (paresis of the limbs and urinary bladder, diminution or absence of reflexes) which is never observed in rachitic curvature of the spine.

In cases of affection of the hip and knee joints involving destruction of the articular ends of the bones the limbs are shortened for life.

Affections of the wrist, ankle and other joints cause lesser deformation and crippling.

Tuberculosis of the bones and joints is a very protracted disease; diagnosed in good time and properly treated it ends favourably, but sometimes, in cases of a very protracted course of the disease and continuous pyorrhea, it causes degeneration of internal organs (amyloid degeneration of the liver and kidneys) and results in death.

In tuberculous affection of the phalanges of the fingers puffiness and reddening of the affected parts (spina ventosa or tuberculous dactylitis) are observed.

TUBERCULOSIS CUTIS

Tuberculous lesions of the skin are sometimes the first symptoms of tuberculous infection in children.

So-called *tuberculides*, which are usually quite soon followed by manifestations of affections of some organ, are a kind of skin reaction to a latent tuberculous process. Tuberculides are of great diagnostic value.

In recent years, histological sections and grafting patches of skin with tuberculides to animals helped to establish the tuberculous nature of these skin lesions.

Tuberculides appear on the skin as small (the size of a pinhead or somewhat bigger) red papules with a small crater-like depression in the centre.

In some cases there are but a few of them (3-4), in others they number several dozen. They eventually heal with a residual bright scar. Tuberculides are often seen in nurslings affected with miliary tuberculosis or tuberculous meningitis; in older children they occur less frequently.

Scrofuloderma is a frequent tuberculous lesion observed almost exclusively in children. Large painless nodes appear under the skin; in the beginning they are movable, but then gradually grow into the skin. The nodes vary in size from a pea to a hazel-nut, and resemble deep-seated furuncles. Sometimes they are resorbed but more often soften in the middle and rupture, forming an ulcer with deeply undermined, *thin* (as distinct from a syphilitic ulcer) edges. The skin surrounding the ulcer is violet-reddish. Sometimes tubercle bacilli are found in the purulent discharge.

Healing is slow, residual reddish scars with strands and patches of intact skin remaining on the skin.

Lichen scrofulosus (tuberculosis lichenoides) is marked by small (pinhead-sized) yellow-reddish papules which appear in large numbers most often on the lateral surfaces of the chest and on the back; they are usually arranged in the form of plaques, several centimetres large, persist for several weeks and disappear completely, without leaving a trace. This disease is usually associated with the forms of tuberculosis which run a favourable course.

Lupus vulgaris is tuberculosis of the skin; it usually begins in childhood (its incidence being 40 per cent in children under twelve years of age and 60 per cent in the period of pubescence); this affection seldom results in death but causes grave, life-long disfiguration.

The disease begins with the formation of papules in the derma usually on the wings of the nose (alae nasi) and on the cheeks. Eventually the papules coalesce, forming subepithelial infiltrates which in a favourable course of the disease may undergo retrogression and form superficial scars with a silky glitter. In other, more frequent, cases the affected surface becomes ulcerated. The process may extend from the surface of the skin (of the face and the wings of the nose) to the mucosa; in this case grave destruction of the nose, mouth and even larynx is observed.

The destructive action of the disease may be prevented by early diagnosis and proper treatment (A. Kisel). Fresh air, sunshine and heliotherapy are very important in the treatment of the affected child.

TUBERCULOSIS OF THE MUCOUS MEMBRANES

Among tuberculous affections of the visible mucous membranes lesions of the conjunctiva (of the eye) are most often observed. The disease is marked by the appearance of so-called phlyctenae—vesicles situated predominantly at the edge of the cornea, and attended with general conjunctivitis with photophobia and epiphora.

Infiltrates similar to those observed on the conjunctiva may appear on the cornea with resultant corneal opacity.

No tubercle bacilli are found in the phlyctenae. However, observations show that phlyctenae, like tuberculides and other skin affections, are often indicative of a tuberculous process developing in other organs and must therefore be regarded as an affection closely connected with tuberculous infection.

GENERALISED FORMS OF TUBERCULOSIS

General miliary tuberculosis. Generalisation of the tuberculous process (its rapid spread to many organs) is characteristic of childhood, especially early childhood (Fig. 31).

Sometimes, the child's organism fails to cope with tuberculous infection from its very onset, the infection is not localised at any one site, tubercles forming in many organs and the child dying of a general tuberculous process.

In other cases this dissemination of infection with the blood flow follows certain acute infections, especially measles, whooping cough, influenza and pneumonia. At this time the child's immunity sharply diminishes and the infection spreads (disseminates) from the old tuberculous focus to many organs, i.e., gives rise to so-called general miliary tuberculosis.

In some cases the onset of the disease sometimes simulates influenza, in others—typhus, the more so since the liver and spleen are enlarged. At this time the Pirquet reaction may be negative as in any severe form of tuberculosis. Unlike typhoid fever, the blood in miliary tuberculosis shows leukocytosis; the Widal test is negative (see *Typhoid Fever*). In some cases tuberculides are seen on the skin, or examination of the fundus oculi reveals tubercles in the mucous membranes which permits of a more exact diagnosis.

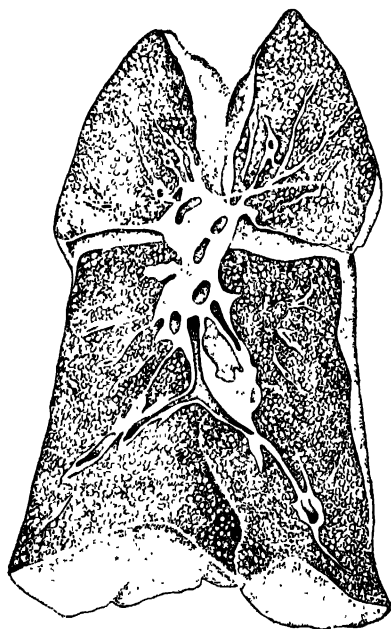


Fig. 31. Miliary tuberculosis

Tuberculous meningitis is one of the manifestations of general miliary tuberculosis; it affects mostly children of younger age and is not infrequent in infants. The pathological changes consist in tubercles appearing on the pia mater and formation of a jelly-like exudate on the base of the brain; eventually acute hydrocephalus (dilatation of cerebral ventricles) develops.

The onset of the disease is not so acute and abrupt as it is in epidemic cerebrospinal meningitis (see *Epidemic Cerebrospinal Meningitis*) or in purulent meningitis; in some cases, mostly in older children, some prodromal symptoms are observed.

About two or three weeks before the appearance of more definite symptoms of meningitis, the affected children become apathetic, run a fever, have headaches

and subsequently vomit; signs of meningeal irritation (Kernig's sign) and occipital rigidity become evident.

To ascertain the diagnosis a spinal puncture is made: in case of tuberculous meningitis the fluid is transparent; only in passing light (through a test tube) it shows, as it were, particles of dust; the protein content is considerably increased, Pandy's test* is definitely positive; allowed to stand in the cold it forms a deli-

* A mixture of 80 to 100 cc of phenol and distilled water is placed in a test tube and one drop of spinal fluid is added. If increased protein is present, a bluish white ring or cloud is formed.

cate film in which tubercle bacilli may often be discovered. The disease lasts 3-4 weeks; formerly it usually ended in death.

Early treatment of tuberculous meningitis with streptomycin and phthivazide now frequently brings about recovery. In such cases acute inflammatory tuberculous meningitis becomes chronic, owing to which the inflammation of meninges ceases, but tuberculosis persists since it affects the whole organism; its treatment as well as prophylactic measures to avoid aggravations must continue for a long time.

DIAGNOSIS OF TUBERCULOSIS IN CHILDREN

The anamnesis is very important for diagnosing tuberculosis in children; it is essential to know whether the parents, especially the mother, are affected with tuberculosis.

It is also important to find out whether the child has been in contact, however brief, with friends or relatives affected with active tuberculosis.

To confirm the diagnosis in accordance with the above-described clinical symptoms of a particular affection, roentgenoscopy, examination of the sputum, urine and feces for tubercle bacilli are employed. Special cutaneous reactions for tuberculosis (the Pirquet and Mantoux tests, for the technique see pp. 114-115) should also be performed.

Roentgenoscopy is very important for diagnosing tuberculosis; in some cases tuberculosis may be difficult to diagnose and roentgenoscopy is often the decisive factor in diagnosing the disease.

Fluorography has been used in recent years to detect pulmonary tuberculosis in children. Fluorography is a combination of fluoroscopy and photography whereby a photograph of small size is made of the fluoroscopic image. The advantage of this method of examination is that it permits of examining many children in a short period of time (100 and more children per hour), i.e., masses of children; the low cost of fluorographic pictures makes it possible to examine the same children more frequently, which is an important factor in dynamic observation.

In addition to the fluorographic method a new method of sectional radiography is now used to examine the lungs; by this method it is possible to determine the localisation and nature of a tuberculous focus more accurately. This is done with a special apparatus known as a tomograph. The apparatus was recently reconstructed by being combined with a fluorograph; the tomofluorograph permits of making as many pictures as are made by the fluorographic method, making them simultaneously of many plane sections.

PREVENTION OF TUBERCULOSIS

A proper regimen, including plenty of fresh air, and organisation of special forest schools, or special classes for weak children, are all important measures in the prevention and public control of tuberculosis in children.

Nor must such prophylactic measures as safeguarding the child against becoming infected from the tuberculous people surrounding him be neglected. It is sometimes expedient temporarily to separate the child from the family (at the time the tuberculous process is particularly active in the mother or other relatives).

In the U.S.S.R. the basic institutions for control of tuberculosis are dispensaries for tuberculous patients with special children's departments.

The dispensaries for tuberculous patients not only administer aid to affected children, but also carry out a number of social and prophylactic measures (early detection of tuberculosis, health education work at children's institutions, at homes, etc.). The system of dispensaries for tuberculous patients includes a number of auxiliary children's institutions (daytime and night sanatoriums, play-grounds).

To prevent active development of the tuberculous process in the child it is necessary to safeguard the child in his early age (up to three years) against infection with measles, whooping cough and pneumonia which are sometimes followed by activation of the tuberculous process and development of grave forms of tuberculosis (miliary tuberculosis, tuberculous meningitis).

Specific prophylaxis. Prophylactic inoculation with Calmette's vaccine against tuberculosis has gained currency in France since 1921. Calmette's method consists in the following: children, mainly those born of tuberculous mothers, are administered, during the first ten days of life, a culture of attenuated human tubercle bacilli per os three times (on the 3rd, 5th and 7th or on the 4th, 6th and 8th day of life). This vaccine is also called the BCG vaccine—*Bacillus Calmette-Guérin*—after the scientists who have prepared and offered it for vaccination. A single dose contains 400,000,000 BCG given with human milk.

In 1925 this method of immunisation was made compulsory in the U.S.S.R.

All newborn infants with no indications against vaccination are inoculated.

The indications against vaccination are as follows: 1) pyrexia above 37.5°; 2) persistent regurgitation; 3) marked dyspeptic dis-

orders; 4) diseases affecting the general condition of the infant (marked skin abscesses, phlegmons, otitis, influenza, pneumonia, etc.).

Observations have demonstrated the harmlessness and effectiveness of these vaccinations. General mortality among vaccinated infants born of tuberculous mothers, children of tuberculous families is only half that of unvaccinated children; according to other data it is but one-third or one-fourth that of unvaccinated children. Inasmuch as vaccination confers only relative immunity, administration of the BCG vaccine by no means obviates the necessity of carrying out general tuberculosis control measures.

It should be remembered that the child acquires even this relative immunity not earlier than 5-6 weeks after the vaccination and the children must, therefore, be thoroughly protected against mass infection by tubercle bacilli.

Since Calmette's vaccination confers only temporary immunity to tuberculosis, children of all ages should be revaccinated. The first revaccination is performed between one and three years of age, the second—between four and seven, the third—between eight and twelve, and the fourth after thirteen years of age.

All healthy children and adolescents with a negative tuberculin reaction, as well as those in whom a thorough clinical examination, including roentgenoscopy, revealed no tuberculous lesions or lesions which may warrant the assumption of tuberculosis, must be revaccinated. Children of tuberculous families with no evident signs of tuberculosis should be removed from their surroundings for one month before and six weeks after revaccination.

Infants, who for some reason failed to be vaccinated in the maternity home, should be vaccinated in an infant health centre or polyclinic during the first two months of their life.

Vaccination of infants and children of preschool age is performed per os or by scarification, while that of school children and adolescents—only by the cutaneous method with a specially prepared vaccine.

TREATMENT OF TUBERCULOSIS

Not all forms of children's tuberculosis necessitate treatment in hospitals; many tuberculous children (chronic tuberculous intoxication, certain skin and bone affections) may be treated in dispensaries, children's polyclinics and infant health centres. Children

with marked chronic tuberculous intoxication must be periodically examined in dispensaries, and placed in forest schools or daytime sanatoriums.

In some cases of localised tuberculosis (pulmonary tuberculosis, tuberculosis of the intestines or bones) the child must for some time be hospitalised or placed in a sanatorium.

Several new preparations have of late been used for treating tuberculosis. As was already mentioned, streptomycin is used in the treatment of tuberculous meningitis. Observations have shown streptomycin to produce a favourable effect also in other forms of tuberculosis: tuberculosis of the larynx, miliary tuberculosis, all fresh infiltrative processes of all localisations and tuberculous lesions of the kidney. In tuberculous meningitis streptomycin is administered intramuscularly, but mainly into the spinal canal, while in other forms of tuberculosis it is administered only intramuscularly.

Soviet scientists have developed a new preparation for treating tuberculosis—paraaminosalicylic acid—which produces a favourable effect in tuberculosis of the lungs, larynx, skin, mucous membranes and urethra. Its total daily dose is 0.25 per 1 kg of the child's body weight; it is divided into four portions and is administered four times a day; the treatment lasts several months, depending upon the gravity of the case. Phthivazide is also effectively used in the following doses: infants between six months and one year of age are given 0.05 g three times a day, children between one and three years of age—0.1 g three times a day, and children from seven to fourteen years of age—0.3 g six times a day. The total dose of phthivazide for the course of treatment lasting from two to two and a half months is 30-40-50 g.

Except the grave and disseminated forms (which are not so frequent) tuberculosis in children runs a rather favourable course. The child's organism resists the tuberculous infection well and it is only necessary to help it by maintaining adequate living conditions and a rational regimen.

Sunshine and fresh air are potent agents in the treatment of tuberculosis. In connection with this the treatment of children in TB sanatoriums is valuable mainly because the children are kept outdoors for the greater part of the day all the year round. Thus, in combination with a proper diet, it is often possible to obtain very good results even in severe tuberculous cases.

It should be added that there is no need sending the affected child to southern sanatorium for treatment, since the treatment

is more effective under the climatic conditions to which the child is accustomed.

Cod-liver oil is a very valuable remedy administered per os, and the child should receive it all the year round (in summer cod-liver oil should be kept in a cold place).

Children with tuberculosis of bones and joints should be given special orthopedic and surgical treatment.

Specific preparations (tuberculin) have not gained currency because of their inefficacy.

Artificial pneumothorax is induced in cases of pulmonary tuberculosis both in children and infants.

In treating lupus vulgaris Soviet medicine effectively uses vitamin D₂ which is administered in an alcohol solution twice a day, at breakfast and dinner (several drops on a slice of bread, and not with water); the daily dose is 50,000-75,000 u, the course of treatment lasting from four to six months, depending upon the severity of the affection.

Observations have proved the effectiveness of vitamin D₂ even in protracted cases of lupus vulgaris.

SYPHILIS

Syphilis in children may be congenital or acquired.

ACQUIRED SYPHILIS

Before the Revolution *acquired* syphilis was a widespread disease among the poor sections of the population who lived under very difficult material and housing conditions.

Syphilis was particularly widespread among the backward and uncultured nationalities of the outlying districts of tsarist Russia. Now this form of the disease is very rarely observed in children.

In its clinical manifestations acquired syphilis of children differs from that of adults. Children acquire syphilis mainly *asexually* (through kissing, common utensils, household articles, sleeping in the same bed with syphilitics).

One of the clinical distinctions of acquired syphilis in children from that of adults is the presence of the primary chancre on the cheeks, lips (mainly lower lip), tongue, tonsils and oral mucosa.

The secondary period is marked by a skin eruption, for the most part macular or papular.

The tertiary period develops after the sixth or seventh years of the infection and is characterised by lesions of bones (periostitis, gumma), general anemia, and in some cases lesions of the central nervous system (cerebral syphilis, meningitis).

CONGENITAL SYPHILIS

Congenital syphilis plays a more important role in children's pathology than acquired syphilis.

Etiology and pathogenesis. Syphilis is an infectious disease; its causative agent is the *Treponema pallidum*; the organism is called pale (pallidum) because it poorly stains with aniline dyes. The syphilis microbe is extremely unstable and does not survive long outside the human body.

Congenital syphilis in its acute stage is contagious for the surrounding people: fissures at the corners of the mouth, the nasal discharge and moist papules contain great numbers of the causative agent.

In the peripheral blood the *Treponema pallidum* is found only in newborn infants affected with syphilis; their presence in the blood of older children is rare.

The causative agent is transmitted to the fetus from the affected mother through the placenta.

Transmission of the disease by the father at the time of fertilisation without infecting the mother is not considered feasible; in such cases the father first infects his wife, the mother of his future child (she may have latent syphilis) and she, in her turn, transmits the infection to the child through the placenta. As a rule, children born of syphilitic mothers are also affected (up to 97 per cent), and only a small percentage of children of affected mothers are born healthy (up to 3 per cent).

Not infrequently, the pregnancy of syphilitic women does not reach term and spontaneous abortion of the affected fetus occurs. Several pregnancies resulting in repeated abortions indicate considerable virulence of the infection and are most often observed in women with the fresh forms of syphilis. Eventually the infection weakens, stillborn children are produced at term, then live babies who also die soon are born, and, lastly, seemingly normal and healthy babies appear, but subsequently manifest symptoms of syphilis.

Unlike acquired syphilis, congenital syphilis does not produce the primary lesion or chancre (hard chancre). The infection gains entrance into the organism through the blood and the primary stage of the disease is of the character of septicemia which begins during intrauterine life. The internal organs are the first to be affected (visceral syphilis); skin lesions are observed somewhat later. This phase of the disease in its purest form is seen in the infected fetus when the stillbirth occurs in the sixth or seventh

month of pregnancy. Examination of such fetuses reveals that all organs are infected and contain great numbers of the *Treponema pallidum*; stillborn babies may also have skin lesions; live babies born at term may develop the first signs of the disease even at birth, but in the overwhelming majority of cases these signs become evident during the first three months and, less frequently, during the subsequent months of infancy.

There may be cases of congenital syphilis in which clinically distinct symptoms are observed only in older children and, in rare cases, even in adults, usually in the form of tertiary lesions (gummas, periostitis). This is the so-called late syphilis. It is possible that in some cases of late syphilis its primary symptoms were overlooked in early infancy.

CLINICAL MANIFESTATIONS OF CONGENITAL SYPHILIS IN EARLY CHILDHOOD

Skin lesions. Lesions of skin are one of the most frequent manifestations of congenital syphilis, the *Treponema pallidum* being easily found in up to 90 per cent of the skin eruptions. The eruptions are not infrequently accompanied by an elevation of the temperature; children are very restless at this period.



Fig. 32. Syphilitic pemphigus

Almost one-third of the cases with skin eruption is followed by relapses; energetic treatment reduces the number of relapses considerably. The skin eruptions vary with the character of the affection.

Syphilitic pemphigus (Fig. 32) is often evident even at birth or may appear soon after birth.

The eruption consists of vesicles, from a pea to a cherry in size, containing first serous and then turbid purulent fluid with large numbers of the *Treponema pallidum*. The eruption is localised predominantly on the extremities, especially on the soles of the feet and on the palms, sometimes on the shins, forearms and, more rarely, on the face and trunk.

There is no such typical localisation in ordinary nonsyphilitic pemphigus (pemphigus simplex), and the vesicles are scattered over various parts of the body and head. Syphilis aggravated by manifestations of pemphigus produces high mortality.

Diffuse skin affection—infiltration—is quite characteristic of syphilis; the eruption is localised mainly on the soles of the feet, palms, face, hairy part of the head, and, more rarely, on various parts of the body. This is the most frequent form of skin eruption (up to 60 per cent). It is not observed before the end of the first month of life; it appears most frequently during the second month of life and, as an exception, during the second half of the year. This kind of infiltration produces a characteristic lustre on the palms and the soles of the feet: the skin appears, as it were, polished and reddish. This is followed by desquamation, more often scaly desquamation and, if the hairy part of the head is affected, by characteristic alopecia in patches of irregular form and size. The lips and adjacent skin develop diffuse indurations, the lips becoming edematous, thickened and covered with radial bleeding fissures.

A *papular eruption* occurs not infrequently; it appears later (in the 4th, 6th or 8th week after birth), the papules localising on the face, extremities and buttocks.

The eruption consists of small pinkish spots; sometimes the spots may be brownish-red with a peculiar lustre. The eruption has many varieties; it is often polymorphic (maculopapular) and is sometimes accompanied by other skin lesions).

On the face the papules not infrequently develop into pustules, which open and form ulcers subsequently covered with crusts (papulopustular eruption).

Affections of nails and hair. In some cases of congenital syphilis the nails are tubular and striated longitudinally and transversely; in some cases fissures appear, and sometimes detachment of the nail is observed. Affection of the skin on the head gives rise to thinning of the hair and partial alopecia; sometimes the newborn affected with congenital syphilis have no eyebrows and their eyelashes are irregularly arranged.

Affections of the mucosa. Syphilitic rhinitis is one of the most frequent affections of the nasal mucosa. It often starts during intrauterine life. Rhinitis is one of the earliest and most frequent symptoms of congenital syphilis (up to 70 per cent); most often it makes its appearance during the first four weeks of life and is characterised by difficulties in nasal breathing and a mucopurulent and sometimes sanious discharge.

The affection of the nose is a chronic process, sometimes very protracted; in some cases the cartilage and the bony part of the nose are affected, and scarring, contraction of pliable parts and deformation of the nose (saddle nose) occur. However, this process does not destroy the bony or cartilaginous part of the nose, and in such cases necroses are very rare.

The mucous membranes of other organs—larynx, mouth, the fauces and anus—are affected less frequently. In lesions of the larynx a hoarse voice, aphonia and, in rare cases, stenosis may be observed. Superficial ulceration in the form of separate plaques is sometimes found on the oral and faucial mucosa.

Affections of internal organs constitute an important part of the manifestations of syphilis; they may appear during intrauterine life when skin lesions are not yet perceptible.

Enlargement of the spleen is the most frequent and earliest affection (80 per cent of all cases). The spleen is dense and protrudes from under the ribs. Anatomically it contains enlarged cellular elements; examination of the pulp reveals a considerable number of the *Treponema pallidum*.

The liver is affected as frequently: it is dense, enlarged; sometimes the newborn have jaundice of a specific character. The anatomic changes in the liver are characterised by diffuse cellular infiltration; older children sometimes have connective tissue hyperplasia which clinically produces a picture of liver cirrhosis.

Lesions of the kidneys are clinically manifested by albumin, casts and cellular elements in the urine; post-mortem examinations often reveal diffuse affection of the connective tissue.

The changes in the cardiovascular system are manifested mainly in lesions of the vascular intima (endarteritis).

The symptoms vary with the organ whose vessels are affected. Lesions of heart vessels are characterised by disturbances in respiration and cyanosis; in lesions of cerebral vessels symptoms of encephalitis are observed.

Pulmonary affections in congenital syphilis usually begin during intrauterine life; characteristic proliferation of the pulmonary connective tissue (pneumonia alga) is rarely observed in children born alive; though children affected with syphilis often die of pneumonia (up to 25 per cent), this type of pneumonia is of a nonspecific character.

Gastrointestinal disorders (vomiting, diarrhea) are not infrequently observed in children with congenital syphilis, but they are not always of a specific character and are mostly ordinary dyspepsias of early childhood.

Affection of the testes. Congenital syphilis is not infrequently attended with affection of the testes, mainly hydrocele, with the testes dense and enlarged.

Affections of lymph nodes. In syphilis the lymph nodes are often enlarged, but since they may also be enlarged in other diseases (tuberculosis), this symptom cannot serve as sufficient ground for diagnosing syphilis; postmortem examinations do not reveal any specific changes in the lymph nodes of children affected with syphilis.

The greatest importance is attached to enlargement of the ulnar lymph nodes; with other symptoms of syphilis in evidence it may be given consideration in confirming the diagnosis.

Bone affections. Involvement of the bones in early congenital syphilis—osteochondritis—is very frequent. It is due to the fact that during the period of vigorous growth and formation of bone and cartilage cells, particularly in places where this growth is the most intense (borderline between the diaphysis and epiphysis of the long bones), there is a considerable influx of blood which provides the causative agents with a favourable medium. The inflammation may result in separation of the epiphysis from the diaphysis and the affected limb seems to be paralysed; the arm hangs limply and the leg is drawn to the abdomen. No changes are usually observed in the nervous system and the paralysis of the extremities is essentially false (Parrot's pseudoparalysis). In addition to the bone, the periosteum is also involved in the process; the surface of the bone is thickened, mostly unevenly; sometimes hyperostoses are formed on the cranial bones.

Osteosclerosis is a frequent phenomenon in congenital syphilis; sometimes the process spreads to the spongy substance and the whole bone becomes dense and compact which is very clearly seen in roentgenograms.

Gummas in the bone of children affected with congenital syphilis are found infrequently; only in late congenital syphilis gummatous periostitis, osteitis and osteomyelitis are sometimes observed.

Affections of the nervous system. Hydrocephalus of syphilitic origin resulting from lesions of meninges and cerebral vessels is sometimes observed in babies during the first month of life; it may be congenital.

The continuous (day and night) groundless crying of children affected with congenital syphilis is considered due to irritation of the meninges.

Affections of the eyes. Of the sense organs the eyes are most often affected in syphilis (mainly the retina and cornea).

Syphilitic keratitis is often observed in children of 3-4 years of age; sometimes it occurs earlier—in infants and even in the new-

born. If the child is in poor general condition, keratitis may result in keratomalacia (softening of the cornea).

Anemia in syphilis. Anemia, sometimes strongly pronounced, is not infrequently observed in congenital syphilis; this anemia does not essentially differ from secondary anemia in other diseases and is connected with general disorders of nutrition.

DIAGNOSIS OF CONGENITAL SYPHILIS

Diagnosis of congenital syphilis presents no difficulties if all aforementioned affections of the different organs are sufficiently evident, but it becomes difficult if the characteristic symptoms of syphilis are few or if they are but feebly marked. In such cases only a careful anamnesis is very helpful in diagnosing the disease.

Sometimes the clinical picture of congenital syphilis does not appear at once; a child with no symptoms of syphilis may be born of syphilitic parents.

Since the percentage of healthy children born of syphilitic parents is extremely low, it is best in such cases to overestimate some inconspicuous and vague symptom of congenital syphilis and regard it as evidence of the disease than wait for the picture of the disease to become clearer. The earlier the treatment begins the more hopeful the expectations.

To confirm the diagnosis of syphilis, Wassermann's test is performed; it should be noted, however, that whereas a positive reaction in some measure warrants the assumption of syphilis, a negative reaction by no means denies it. It should be remembered that Wassermann's reaction may be positive in certain other diseases (scarlet fever in its eruption stage, measles in its prodromal stage, malaria and other protozoan diseases); Wassermann's test must be repeated, thorough clinical investigation made and the anamnesis obtained.

In congenital syphilis the prognosis depends upon the gravity of infection and the extent of affection of the different organs. Usually, an early manifestation of syphilis in a newborn attests the severity of the affection and in such cases the prognosis is poorer than in older children.

Artificial feeding and poor living conditions considerably worsen the prognosis. Energetic and early treatment and adequate hygienic conditions sharply reduce mortality.

PREVENTION AND TREATMENT

Syphilis affects the child during intrauterine life, for which reason prevention and treatment of the disease should begin before the child is born.

Syphilitics should be steadfastly treated before marriage. They must not be allowed to marry unless they have undergone 3-4 combined courses of treatment. Moreover, a pregnant woman affected with syphilis must undergo an additional course of vigorous treatment during pregnancy.

Extensive public measures, including dispensary service, keeping records and treatment of syphilitics, considerably decrease the spread of congenital syphilis and consequently lead to its complete eradication.

Affected children are now given combined treatment, preparations of arsenic—novarsenol (neoarsphenamine) and myarsenol (sulfarsphenamine)—mercury (mercuric chloride, mercury cyanide) and bismuth (biochinol) being the main components of the combined treatment of syphilis. There are several types of syphilitic treatment, each essentially consisting of several courses (from four to six) with intervals of 1.5-2 months between the first and second, the second and third, and the third and fourth courses, six months between the fourth and fifth, and two months between the fifth and sixth courses.

Each course consists of intravenous administration of novarsenol or intramuscular injection of myarsenol, which alternate with intramuscular injections of preparations of bismuth (biochinol) and mercury (1 per cent solution of mercury bichloride or mercuric oxycyanide).

There are usually 10-12 infusions or injections of novarsenol or myarsenol and 12-15 of mercury or bismuth preparations.

Treatment with novarsenol or myarsenol is necessarily administered at 4-5-day intervals; the intervals between injections of bismuth or mercury are 1-2 days; usually bismuth injections are administered in the first course of treatment and mercury—in the second; the subsequent alternation is similar. Thus, in a six-course treatment the patient receives six courses of novarsenol or myarsenol and three courses of bismuth and mercury.

Wassermann's test is performed before and after each course of treatment. To simplify the treatment it is now recommended that children past three months of age should be administered the aforesaid preparations not alternately, on different days, but simultaneously, the same day. Novarsenol or myarsenol are the

first to be injected, and half an hour later (if there are no side effects)—biochinol or mercury.

The preparations are administered simultaneously in cases in which the patient cannot, for some reason, visit the clinic frequently; such patients are required to visit the clinic once in five or six days, i.e., to make a total of 9-10 visits.

The dose of antisyphilitic preparations is calculated mainly on the basis of the child's body weight and must be strictly individual with due regard for the child's body weight and his general condition.

Dosage of Antisyphilitic Preparations

Biochinol per 1 kg of body weight	Novarsenol, myarsenol per 1 kg of body weight	Mercury
0.01-0.15 g but not more 1.0 g for infants up to 2 years of age 1.5 g for children of 2-5 years of age 2.0 g for children of 5-15 years of age	0.03 g—up to 1 year of age 0.02 g—up to 2 years of age 0.015-0.01 g—past 2 years of age, single dose not exceeding: 0.15 g for infants under 3 months of age 0.2 g for children aged from 3 months to 5 years; 0.25 g for children aged from 5 to 10 years; 0.3 g for children from 10 to 15 years of age	1. Injections of 1% solution of mercury bichloride or mercuric oxycyanate in a dose of 0.1 ml of solution per 1 kg of body weight, but not more than 0.3 for infants under 3 months, 0.75 for infants between 3 months and 2 years of age, 1.0 for children over 2 years of age. 2. Inunction of mercuric sulfide ointment into the skin for 20 minutes calculated on the basis of 0.01 per 1 kg of body weight but not more than: 1.0 up to 1 year of age 2.0 between 1-5 years of age 3.0 between 5-15 years of age

Children can tolerate much larger doses than do adults, and the younger the child the larger may be the single dose calculated per 1 kg of body weight within the above-mentioned limits, although it must not exceed the definite maximum single dose for the given age. During the first days of treatment the dose is much smaller (one-half to one-third the maximum dose); then it is gradually

increased until it reaches the maximum after 3-4 injections and is retained to the end of the course of treatment.

Penicillin is being used of late in the treatment of syphilis; it is especially indicated in affections of internal organs and in complications by secondary infections. It is administered intramuscularly in doses of 10,000-30,000 u per 1 kg of body weight a day, 3-4 injections in the course of 10-15 days; the doses are gradually increased: 5,000 u per 1 kg of body weight the first day, 10,000 u the second day, etc. During the course of treatment, nurslings receive from 500,000 to 1,500,000 u, children from one to 3-4 years of age—1,500,000-2,000,000 u; older children should be administered 2,000,000-2,500,000 u of penicillin.

Penicillin treatment should be supplemented by four courses of above-mentioned combined treatment with novarsenol (myarsenol), mercury and bismuth preparations.

MALARIA

Etiology and pathogenesis. The pathogenic agent belongs to the Protozoa and is called Plasmodium; three species of it are encountered in man, each of them causing a definite type of fever attacks which alternate with apyretic periods. Thus the Plasmodium vivax causes tertian malaria. The Plasmodium malariae causes quartan malaria and the Plasmodium immaculatum—tropical malaria. All these species of the causative agent of malaria also occur in children suffering from malaria.

The disease is transmitted by the anopheles mosquito. In areas with a climate warm enough for the mosquitoes to multiply and abounding in swamps the disease may become widespread provided there are people infected with malaria. The disease particularly affects children, the incidence among them sometimes reaching almost 100 per cent.

In addition to being infected by the mosquitoes, infants may suffer from congenital malaria, when the Plasmodium is transmitted to the child by its mother during intrauterine life. The affection becomes evident rather soon after birth; in some cases malaria in the mother may be the cause of abortion or the birth of a premature child.

Pathologic anatomy. The pathologic changes caused by malaria are mainly changes in the blood (destruction of erythrocytes) and postmortem examinations reveal a certain measure of anemia and special pigmentation conditioned by the destruction of erythrocytes in many internal organs (spleen, liver, pancreas, bone marrow, brain).

The changes in the spleen are of special significance: it is enlarged, particularly during acute attacks of the disease; the capsule of the considerably enlarged spleen is tense and in some, very rare cases, to be sure, it may even rupture. Sometimes, autopsies of the malarial spleen reveal necrotic foci

(infarctions); in cases of multiple foci a peculiar flecked spleen is observed. Examination of a pulp smear reveals numerous malarial parasites and a lot of pigment deposited in the form of tiny lumps. In chronic malaria the spleen is particularly dense.

In malaria the liver is also not infrequently enlarged; its pigmentation is particularly marked and a section of the liver looks slate-grey or chocolate-coloured.

It is also necessary to mention the changes in the bone marrow; a large amount of deposited pigment and numerous malarial parasites in the form of gametes (reproductive cells) are found in the bone marrow.

Changes in the brain are sometimes observed in grave cases of tropical malaria; the brain is smoke-coloured (especially the grey matter). Microscopic examination of the brain reveals general development of stasis; an abundance of Plasmodia and pigmentation are found in the erythrocytes.

CLINICAL PICTURE AND FORMS OF THE DISEASE

The incubation period in malaria is from 10 to 15 days. However, more time (several weeks and even months) may elapse between the moment of the infection and the attacks of fever.

Sometimes the first attacks of malaria appear in spring when there are no mosquitoes and there is no possibility of infection. Some of these cases are apparently not primary infections but "springtime" relapses.

A typical attack of malaria runs a cyclic course and consists of the following stages: chills, period of high fever and sweating. These stages follow each other in the aforesaid sequence.

The *chills* stage in malaria is very characteristic; sometimes it is feebly marked; in other cases it is very intense ("paroxysmal chills"). The condition is marked by headaches and nausea which sometimes results in vomiting; the skin is dry and cold to touch.

The chills are followed by the second stage—the period of high fever which sometimes reaches 41°; during this stage the patient has headaches and nausea again and not infrequently pain in the abdomen and in the region of the spleen.

With the onset of the third, or *sweating*, period the temperature drops, the skin becomes moist; the sweating is usually profuse; all unpleasant subjective sensations disappear and the patient falls asleep.

The attacks last from 4 to 48 hours.

Protracted attacks are due mainly to long high fever stages; most of the attacks occur in the forenoon. Malaria is characterised by a transition of the highest fever from the evening hours to daytime and early morning hours. A considerable fall in temperature below normal is also characteristic of the disease.

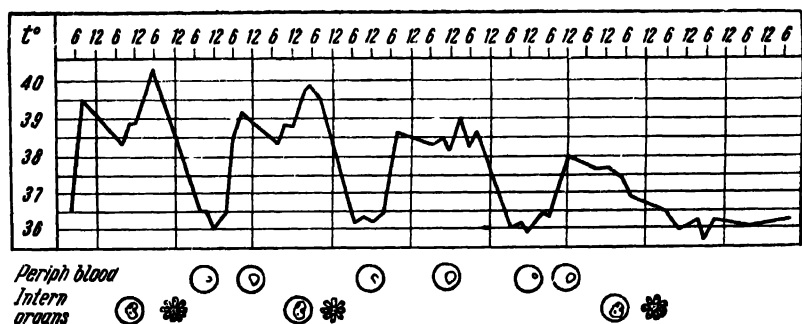
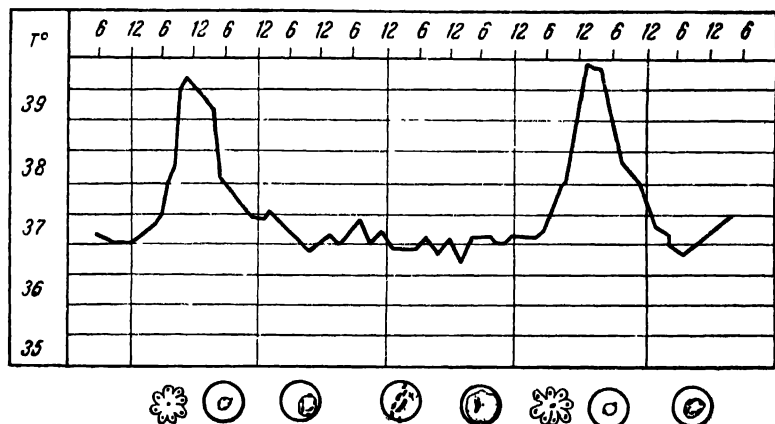
The temperature curve varies with the type of parasite; in tertian malaria the attacks occur every other day or, to be exact, each attack begins on the third day after the beginning of the previous attack (benign tertian malaria) (Fig. 34), but there is a form of malaria also caused by the *Plasmodium vivax*, in which the attacks occur daily (quotidian malaria). The former type is caused by one invasion of parasites in the blood; the latter type is usually caused by two concomitant infections 24 hours apart.

Fig. 33. Temperature curve in quartan malaria

In tropical malaria high fever persists for two or more days. Only by carefully watching the temperature (taking it every two hours) is it possible to detect its maximal elevation on definite days and at definite hours: the temperature curve shows a number of waves with a short period of normal temperature (Fig. 35).

The course of the malarial attack depends upon the life cycle of the Plasmodium.

Blood tests have made it possible to discover different generations of the parasite in the blood at different stages of the attack.



the parasite (minor rings) are observed; at the height of the attack minor and major rings predominate; during the third stage of the disease, when the temperature begins to fall and sweat appears, major rings with considerably thickened bodies are observed. During apyrexia mature forms are observed, and before the beginning of the next attack disintegrating forms (ringlike annular bodies) reappear in the blood. This is the life cycle of the Plasmodium in the blood of tertian and quartan malaria patients.

In tropical malaria the life-cycle of the parasite is not completed in the blood: the phase of the ringlike annular bodies and their disintegration take place in the internal organs; at all stages of the disease only ringlike annular bodies and mature crescent-shaped forms may be found in the blood.

During apyrexia the patient recovers his strength and feels as though he were well again. But, if the paroxysms follow each other for a long time, anemia, characteristic of malaria, develops: the skin becomes pale, sometimes with a slight icteric tint, and the patient grows increasingly weaker; sometimes cardiac murmur and edemas appear. Eventually the fever paroxysms grow weaker, the temperature does not reach the previous level, the chills and sweating cease and a self-cure seems to take place. Subsequently, however, relapses not infrequently occur.

Sometimes the intervals between attacks are very short, relapses follow each other in rapid succession, and chronic malaria sets in, emaciating the patient and producing a picture of acute cachexia and anemia.

Proper treatment sharply alters the course of the disease: sometimes the attacks very abruptly cease and the patient recovers, although in some cases relapses are observed even after vigorous treatment.

In addition to the changes in the blood in which, besides the specific parasites, characteristic malarial leukopenia is found, there are also changes in the internal organs. Of these, a considerable enlargement of the spleen and sometimes also of the liver comes to the fore. During a prolonged chronic malarial process the size of the spleen changes: it enlarges considerably at the time of attack and perceptibly contracts, sometimes to normal size, in the intervals between attacks.

The form of malaria which runs a malignant and most severe course is tropical malaria which sometimes exhibits symptoms of meningitis or encephalitis. The so-called *malarial coma* occurs mainly in tropical malaria. The autopsies of patients who died in a comatose state reveal a considerable accumulation of parasites in the cerebral capillaries, and in some cases cerebral hemorrhages.

In other cases, in constant malarial areas of tropical countries, the children exhibit malarial cachexia, when a tendency to hemorrhages, edemas and diarrhea is added to the signs of grave anemia and deep changes in the liver and spleen. Many organs undergo pigmentation and some of them (kidneys, spleen, intestines) are not infrequently affected with amyloidosis.

Malarial cachexia is encountered in areas with unfavourable living conditions. Children are the first to die of it, while adults, who survived malaria in childhood, acquire a certain immunity to the disease.

The *clinical picture* of malaria in very young children differs from that in older children.

Congenital malaria has not been adequately described. Many authors still dispute the possibility of transmitting malaria in utero and consider the placenta a barrier for the parasite.

Of late, however, quite a few cases of fully proved congenital malaria have been described; in these cases the children had attacks on the second or third day of life, with the parasites found in their blood. But even when the attacks do not occur during the first days of life and take place several weeks (2-3) later, the possibility of congenital malaria is not excluded.

There have been cases when the first symptoms of the disease in the newborn manifested themselves only in slight periodic elevations of temperature, anemia and cachectic failure to gain weight; in some cases this was observed even *when the spleen was not enlarged*. In these cases the results of blood tests were at first negative. Subsequently, however, more thorough and persistent tests revealed the presence of the parasites in the blood. It is quite possible that in congenital malaria or during long intervals between attacks the malarial *Plasmodium* does not appear in the peripheral blood and some provocative conditions are apparently necessary for it to penetrate into the peripheral circulation. In such cases a careful anamnesis provides valuable indications as to the malarial nature of the disease.

Attack of malaria in very young children (under two years of age)—whether congenital or acquired—often runs an atypical course: instead of the chills sometimes a certain excitement, pallor of the face and, in some cases, convulsions are observed. Vomiting is a frequent phenomenon. Often the attacks do not occur regularly. The temperature curve may be irregular. There is usually no typical sweating (profuse perspiration) and, but for the presence of the parasites which are in the end always found in the blood, other infections might be suspected.

DIAGNOSIS

The diagnosis of malaria is based on 1) clinical data and 2) blood tests.

1. The above-described clinical picture of the disease, in which typical attacks with periodic fever occur on definite days and at definite hours, warrants the diagnosis of malaria even without blood tests. In cases in which daily fever is followed by a rapid fall of the temperature to normal, the affection should be differentiated from septic diseases—tuberculosis, pyelitis and typhoid

fever. A shift of the fever from the evening to the morning hours and the fall of the temperature below normal during apyrexia are characteristic of malaria.

In continued fever, as well as in irregular fever which is sometimes observed in tropical malaria, it is very difficult to diagnose malaria without blood tests. The chronic course of malaria with splenomegaly and progressing anemia sometimes simulates pernicious anemia, leukemia, tuberculosis and many other diseases. In such complicated and doubtful cases the diagnosis can be made only on the basis of the anamnestic data (living in malarial area) and blood tests.

2. An efficient blood test (ordinary smears, thick drop) performed during the attack almost always reveals the presence of the parasites in the blood. Only in mild cases in the beginning of the disease there may be very few parasites in the blood and several tests are required to detect their presence. After an intake of quinine and acrichine (quinacrine) the parasites are more difficult to find.

In cases of suspected malaria, in which no blood tests can be made (absence of a microscope), the patient is given quinine for a period of 3-5 days. Failure to produce a favourable effect will indicate the nonmalarial nature of the disease.

Prognosis. The outcome of malaria in children is favourable in the overwhelming majority of cases provided the disease was diagnosed early and was treated vigorously enough. Only grave forms of tropical malaria in constant malarial areas have been responsible for the very high mortality, among very young children especially.

PROPHYLAXIS

Public prophylaxis consists mainly in extensive sanative measures, i.e., drainage of swamps, mass destruction of the mosquito larvae by petrolising swamps and pools or by dusting them with poisonous substances (Schweinfurth green) from aeroplanes. To destroy mosquito larvae, a special kind of fish—*Gambusia* which feeds upon these larvae—is cultivated in ponds and other water reservoirs.

Organisation of malarial (and tropical) stations which carry on extensive prophylactic, therapeutic and organisational work to control malaria is very helpful. Prevention of malaria is divided into individual preventive measures (netting the premises, screening the beds, etc.) and measures of public prophylaxis (petrolisation, dusting, etc.).

In choosing the site for children's summer camps (Young Pioneers' camps, children's playgrounds) it is necessary to avoid swampy areas and reservoirs in the neighbourhood, because they may be mosquito breeding-grounds.

In malarial areas it is recommended to use quinine or acrichine and bigumal (paludrine) as a prophylactic measure as long as there are people in those areas; administration of these medicines should begin simultaneously with the mass appearance of mosquitoes, but not earlier than July 1 (for those arriving after July 1, from the day of their arrival); acrichine and bigumal should be taken all through the epidemic season in malarial areas and for one month after departure from those areas.

For prophylactic purposes quinine should be taken once a day every other day or daily (in cases of widespread malaria in the given area) in a dose one-third the daily therapeutic dose.

Thus, for a six-year-old child a therapeutic dose of quinine is $0.1 \text{ g} \times 6 = 0.6 \text{ g}$, and a preventive dose is $0.6 \text{ g} : 3 = 0.2 \text{ g}$.

In malarial areas healthy children take acrichine in a dose two-thirds the therapeutic dose once a day for two days in succession with 3-5 day intervals. The intervals are calculated according to epidemiologic conditions (the greater the possibility of infection, the shorter the intervals).

Bigumal is given for the same purpose twice a week in a dose one-third the daily therapeutic dose.

TREATMENT

The most reliable treatment of malaria is treatment with quinine. Quinine is taken by mouth or is injected (intramuscularly, subcutaneously or intravenously); however, quinine injections for very young children should be avoided (because of the frequent formation of abscesses).

The simplest method is to administer quinine per os. It is given in several doses (3-4) per day on the basis of the following calculation: to infants up to one year— 0.01 g a day for every month of their age, but not more than 0.1 g ; to children from one to ten years of age— 0.1 g a day for every year of their age; to children from ten to fifteen— 1.0 g a day; adolescents past fifteen years of age are prescribed adult doses.

The following scheme elaborated by the Moscow tropical Institute is now very widely used in the U.S.S.R.: three days of quinine administration in the aforesaid doses followed by four-day intervals, these cycles continuing for 1-2 months.

Because of its bitterness, in treating very young children quinine is replaced by euquinine (the dose is 50 per cent larger). The treatment of malaria is usually conducted for three seasons, i.e., for a year and a half (for instance, spring, autumn, spring).

In addition to quinine *synthetic preparations*—*acrichine* and *plasmocide* (pamaquine naphthoate) are widely used in the treatment of malaria (Table 14).

Table 14

Acrichine (Quinacrine) and Plasmocide Doses

Age (in years)	Daily acrichine dose taken by mouth (in g)	Acrichine dose injected intramuscularly (4% acrichine solution in ml)	Daily plasmocide dose (in g)
up to 1 year	0.025	0.5-1.0	not prescribed
1-2	0.05	1.0-1.25	0.01
2-4	0.075	1.5-2.0	0.015
4-6	0.1	2.0-3.0	0.02
6-8	0.15	3.0-4.0	0.03
8-12	0.15-0.2	4.0-5.0	0.03-0.04
12-16	0.25	5.0-6.0	0.05
past 16 years	0.3	6.0-7.5	0.06

Children under 4-5 years of age are given acrichine in solution by mouth twice a day. One prescription should contain 10 single doses of solution. Thus, a child one and a half years old is prescribed a solution of 0.25 g of acrichine per 50.0 g of water, one teaspoonful (i.e., 5 ml) twice a day. Acrichine may also be given to children with jam after first powdering the pills, or in the form of sugarcoated acrichine pills. In grave cases of malaria it is recommended to begin the treatment with intramuscular injections of acrichine solutions which even nurslings tolerate well.

Every acrichine powder is washed down with water. In virtue of the profuse perspiration during the hot time of the year urination diminishes and may impede excretion of acrichine; to stimulate urination, it is therefore recommended to give the patients taking acrichine a lot to drink.

The daily dose is given in one intake; a double daily dose is given on the first day, in which case the first cycle is not five but four days; the second and third cycles are three days each; the intervals between the cycles are 7-10 days. Thus, the whole course of treatment is 25-31 days. After a month's interval the course of

treatment is repeated with two-thirds of the usual doses. A similar course of prophylactic treatment should be repeated in spring. Acrichine produces no unpleasant side effects except a yellowish pigmentation of the skin. The preparation is well tolerated by children and is a valuable medicine for malaria.

Inasmuch as acrichine does not act upon gametocytes and destroys only the schizonts of the *Plasmodium*, administration of *plasmocide* is recommended in addition to acrichine during epidemic seasons. It is dispensed only in combination with acrichine in green pills (stained by methylene blue), each pill containing 0.01 g of plasmocide and 0.05 g of acrichine. The maximal daily dose of plasmocide for children from one to two years of age is 0.01 g, i.e., one children's pill; for older children the dose is correspondingly increased.

Plasmocide taken in a dose corresponding to age is well tolerated and does not cause any side or toxic effects, and its combination with acrichine makes it easier to tolerate.

If overdosed, plasmocide produces side effects: headache and epigastric pain, peripheral paralyses, cerebellar symptoms and atrophy of the optic nerve. On the appearance of nervous symptoms the administration of plasmocide should be suspended and the patient's stomach washed out; the child should be given a lot to drink and administered alkalies and caffeine; narcotics must be avoided. Vitamin B₁, nicotinic acid and liver preparations are indicated.

A new antimalarial preparation—bigumal—was recently synthesised in the U.S.S.R.; it is a white, bitter, fine crystalline powder; it is dispensed in white pills of 0.1 g (for adults) or 0.05 g (for children) (Table 15).

Table 15

Bigumal Dosage	
Age in years	Daily dose taken per os in g
Up to 1 year	0.025
1-2	0.05
2-4	0.075
4-6	0.1
6-8	0.15
8-12	0.15-0.2
12-16	0.25

Bigumal enters the blood rapidly and by the end of the first hour after the intake is found in the urine; it is excreted with the urine quicker than acrichine. Children tolerate bigumal well, since it is scarcely toxic; it does not cause leukopenia; a temporary leukocytosis in the peripheral blood and the appearance of immature neutrophils are sometimes observed; there are apparently no contraindications for prescribing bigumal.

The treatment of malaria with bigumal requires one five-day cycle; the treatment of tropical malaria with bigumal is limited to one five-day cycle. In tertian and quartan malaria one-third of the daily dose of bigumal is administered once a week for 3-6 months after the main five-day cycle

MOST IMPORTANT DISEASES OF THE NERVOUS SYSTEM IN CHILDREN

There is no disease that does not in some measure involve the central nervous system. Any pathological process irritates the nerve endings in the affected area. This irritation is transmitted by nerve conductors to nerve centres and evokes a response reaction on the part of the nervous system.

The course of the disease largely depends upon the intensity of the nervous reaction.

Various symptoms of nervous affections may be observed almost in all children's diseases, especially in those running a severe course; these symptoms are most frequently found in infectious diseases. The affections are not stable, however, and usually disappear along with the main disease. In some children's infections the central nervous system may be more seriously affected—from lesions of the cortex to lesions of the peripheral nerves (encephalitis as a result of measles, chickenpox, influenza, etc., and peripheral paralyses in diphtheria).

Infection may also affect the nervous system separately, as it were, and produce a clinical picture of its specific infectious diseases (epidemic cerebrospinal meningitis, poliomyelitis, lethargic encephalitis, tetanus).

Chronic infections (syphilis, tuberculosis) also play a significant role in the etiology of nervous affections in children. In the pathology of the nervous system in children an important part is also played by such factors as intrauterine diseases, injuries sustained at birth and various unfavourable influences sometimes connect-

ed with the diseases of the mother during pregnancy or fetal diseases which affect the formation of the central nervous system.

Some diseases of the nervous system in children (for instance hysteria) are of the nature of functional disorders, in which no organic changes in the central nervous system can be found.

Only the most important and characteristic diseases of the nervous system in children are described in this book.

ENCEPHALITIS (INFLAMMATION OF THE BRAIN)

Encephalitis is most often observed in very young children; it may occur even during intrauterine life. But it is rather difficult to diagnose it in vivo soon after birth and only separate symptoms (paralyses, feeble-mindedness) will suggest some time later the possibility of encephalitis having occurred in utero.

Acute inflammation of the brain may accompany various purulent processes in the brain or its meninges (cerebral abscess, purulent meningitis). In such cases encephalitis is also of a purulent character and is regarded as an affection accompanying the main disease.

However, nonpurulent encephalitis which appears as a result of general infection, or specific cerebral encephalitis is more frequent and more important.

Acute inflammation of the brain—encephalitis—mostly occurs as a sequela of general infection (measles, influenza, chickenpox, and, in rare cases, vaccination); more often encephalitis follows an infection, but sometimes is concomitant with the infection.

Pathologic anatomy. Anatomically, encephalitis is characterised by diffuse or focal lesions mainly of the cerebral cortex or cerebellum; in the initial stages it is marked by multiple punctuate hemorrhages; later cicatricial tissue (sclerosis) may form as a result of these changes; in some cases a cyst and destruction of a definite part of the cerebral substance at the inflamed site are observed.

The *clinical picture* of the disease varies with the anatomical lesions of the particular part of the brain.

Various cerebral lesions, however, may also have common symptoms: as a rule, the onset of the disease is marked by severe cerebral symptoms (convulsive seizures, vomiting, clouded consciousness); not infrequently meningeal symptoms (occipital rigidity, Kernig's sign, red dermography) are also evident. These

symptoms are usually accompanied by considerable elevation of temperature which persists for several days.

The convulsive seizures which are the most characteristic symptom of encephalitis may vary: they may be either general convulsions of the whole body or twitching of separate groups of muscles (one side of the face or body, one upper or one lower extremity). Not infrequently these convulsions are followed by paralysis or paresis of definite groups of muscles in cases in which the inflammation is localised in the motor area of the cerebral cortex. In some cases these paralyse pass rapidly, but in others a picture of stable cerebral infantile paralysis develops and may subsequently be followed by certain mental retardation; severe cases sometimes result in idiocy.

Diagnosis. The diagnosis of encephalitis is based mainly on the above-mentioned symptoms provided they are stable and rather protracted. General cerebral and meningeal symptoms often accompany many children's infections; they do not depend upon inflammatory phenomena in the brain, but are connected with changes in its blood circulation and soon pass. In addition, the child may develop convulsions during various infections at the time the organism is in a special condition known as spasmodophilia.

In the presence of meningeal symptoms a differential diagnosis is made on the basis of data furnished by a lumbar puncture: in cases of meningitis the cerebrospinal fluid is turbid, the amount of protein in it is increased (Pandy's reaction is positive), while in encephalitis the fluid is usually transparent, the amount of protein is normal or slightly increased (Pandy's reaction is negative or weakly positive).

The *prognosis* in every form of encephalitis, even if it does not run a very severe course and there is no paralysis, should be made with caution because focal cerebral lesions not infrequently produce stable changes in the brain which eventually may result in dementia.

Treatment. During the acute stage of the disease rest and application of ice to the head or frequently changed cold compresses to the forehead are recommended. In cases of pyrexia—baths or cold packs, in cases of convulsive seizures—chloral hydrate enemas

Rp. Chlorali hydrati 2.0

Aq. Destill 100.0

MDS 25-50 ml per enema, depending on the child's age

Urotropin is given by mouth, 0.2-0.3 g three times a day. Of late, acute encephalitis and meningoencephalitis have been treated with penicillin (intramuscularly and intralumbally).

In cases of clouded consciousness the child must be fed carefully so that the food may not be inhaled into the respiratory tract with pneumonia developing as a result; if necessary feeding tubes are used.

INFANTILE CEREBRAL PARALYSES

Stable *spastic cerebral paralyses* may develop after encephalitis in accordance with its gravity and localisation. These paralyses may occur not only after the acute course of encephalitis resulting



Fig 36 Spastic hemiplegia



Fig 37 Spastic diplegia

from some infectious disease as is the case of flaccid paralyses developing after the inflammation of the grey matter of the spinal cord (poliomyelitis); they may appear as a sequela of cerebral abscesses, tumours, various cerebral traumas in which the cerebral substance is affected or cerebral hemorrhages take place. Paralyses of this kind may also occur as a result of embolisms or thrombosis in cardiac diseases. In most cases these paralyses are *unilateral*

for only one half of the brain is usually affected. Paralysis may also be caused by some intrauterine lesion (intrauterine paralysis) or result from certain cerebral lesions (hemorrhages) during birth. Paralysis of this etiology are most often *bilateral*. Two forms of spastic cerebral paralysis are therefore distinguished clinically: 1) spastic hemiplegia (Fig. 36) and 2) spastic diplegia (Fig. 37) or Little's disease.

Spastic Hemiplegia

Unilateral spastic paralysis of the arm, leg and facial nerve of the same side is observed in spastic hemiplegia, the lesion of the facial nerve is evident in laughing, crying or grinning when one side of the face becomes distorted; sometimes glassoplegia (when put out, the tongue moves to the unaffected side) and in some cases strabismus are also observed. Paralysis of the extremities produces permanent contracture, the affected arm or leg lagging behind in growth. The tendon reflexes are sharply increased, the Babinsky reflex appears and trophic disturbances (cooling and cyanosis of the skin on the affected side) are evident.

Considerable changes in the cerebral tissue may cause various abnormalities in the character of the child, his mental retardation, sometimes epilepsy, though there have been cases in which the child's mental development remained normal.

The prognosis is poor because children become invalids, especially in cases involving the child's retarded mental development.

Treatment. The treatment is orthopedic and surgical; it is started some time after the nature of paralysis has been established. Until then general restorative treatment—baths, electrification and massage—is administered. Various passive movements are used in orthopedic treatment.

Spastic Diplegia

Spastic diplegia is most frequently caused by hemorrhages into the cerebral cortex occurring during birth.

The initial period of spastic diplegia (Little's disease) is characterised by marked muscular rigidity which affects both sides of the body, especially the lower extremities. Sometimes a picture of seemingly complete torpor is observed. The legs of an affected child suspended from the shoulders cross. Eventually, when the child begins to walk (which happens rather late) this spastic character of the affection of both legs becomes more evident: the child tends to walk more on his toes and crosses his legs. In rare cases rigidity of the upper extremities is also observed and in the most severe cases persistent general rigidity of the whole body may develop.

The child's mental development is usually retarded and sometimes idiocy is observed, especially in cases of microcephaly (abnormal smallness of the head).

The prognosis is as poor as in any cerebral paralysis.

The treatment is the same as in spastic hemiplegia.

EPIDEMIC INFANTILE SPINAL PARALYSIS, ACUTE ANTERIOR POLIOMYELITIS

Etiology. The pathogenic agent is a filtrable virus. According to the latest data, the infection gains entrance through the mucosa of the gastrointestinal tract, as the main and common infection way, and through the nasopharynx. However, droplet infection occurs much less frequently. In the opinion of most authors, from the mucosa of the gastrointestinal tract or the nasopharynx the poliomyelitis virus penetrates through the perineural lymphatics into the central nervous system where it localises itself mainly in the grey matter of the anterior horns of the spinal cord. The virus passes through a porcelain filter (Pasteur-Chamberland filter), the filtrate being pathogenic to experimental animals.

Epidemiology. The disease occurs both in sporadic and epidemic forms. Since 1887 a number of grave epidemics have occurred in Sweden, Norway, the United States of America, Germany. No widespread epidemics were ever observed in the U.S.S.R.

Acute anterior poliomyelitis is primarily a disease of children and even infants, children up to ten years of age accounting for 96 per cent of its incidence. According to the data based on many years of observations, more than 80 per cent of the patients are children under four years of age.

Seasonal flare-ups of the disease are observed at the end of summer and beginning of autumn. Abortive or mild forms of the disease play an important part in its spread and explain why it is scattered and not highly contagious.

Pathologic anatomy and pathogenesis. The changes take place mainly in the grey matter of the spinal cord; however, the grey matter of the brain (cortex and the nuclei of the medulla oblongata) is also involved in no lesser degree. The changes in the white matter are negligible. The involvement of the brain consists of inflammatory changes in the vascular system, degeneration of nerve cells and fibres and glial proliferation. The meninges are also not infrequently involved, for which reason infantile paralysis is in its essence an encephalomeningomyelitis.

Degeneration and atrophy are observed in the muscles affected with paralysis.

Clinical manifestations of the disease depend upon the localisation of processes in the nervous system; flaccid paralyses of a peripheral type are due to affection of the anterior horns; disturbances in swallowing, respiration and cardiac activity are caused by lesions of corresponding nuclei in the medulla oblongata; central paralyses are due to affection of the cerebral cortex; pains are produced by affection of nerve roots and nerve trunks.

Retrogression of paralysis, typical of poliomyelitis, is due to development of an acute reactive inflammation around a small focus which subsequently clears up, sometimes leaving only negligible permanent changes in the motor area.

An attack of the disease confers stable immunity.

Clinical findings. The incubation period is three to ten days. The disease has four periods: fever period (preparalytic), period of paralyzes (paralytic), restoration period and residual period.

Preparalytic period. The disease sets on suddenly and is characterised by fever, headaches and vomiting. Very typical are the signs of hyperesthesia. The child does not allow to touch him and cries when he is touched. Hidrosis and leukopenia are also characteristic of the early period.

There are no special changes in the organs; catarrhs of mucous membranes, catarrhal angina, bronchitis and intestinal disorders are observed. Besides the aforementioned phenomena no changes in the nervous system are found. The temperature remains high for 2-5 days and simulates croupous pneumonia. Within 3-5 days the temperature falls sharply and paralyzes develop.

Paralytic period. Paralyzes develop rapidly and not infrequently involve all four extremities. Characteristic of this period is the fact that paralyzes are most strongly pronounced during the first days of their development and then, after a certain period, stable paralyzes begin to subside.

Paralysis of the *lower extremities* is most often observed in its typical form; the quadriceps femoris and peroneus muscles are paralysed especially frequently (either in one or in both legs); then the *upper extremities* and shoulder girdle, particularly the deltoid muscle, are often affected. The paralysed extremities are immovable, although sometimes the child can produce some movements, for example, move his fingers or toes.

The general character of the lesions is proximal, i.e., the groups of muscles located nearer to the trunk are the first to be affected.

If the quadriceps femoris is paralysed, the raised shank drops limply. In paralysis of the peroneus muscle the foot cannot be extended and hangs lifelessly when the patient is in a sitting position. In paralysis of the shoulder girdle and deltoid muscles the arm hangs motionlessly and cannot be raised to the level of the shoulder. If the muscles of the neck are paralysed, the head hangs forward, backward or sideways. In paralysis of the abdominal muscles the abdomen becomes distended and flabby, sometimes unilaterally. The paralyzes of intercostal muscles and the diaphragm are very grave since they impair respiration.

The affected muscles are flabby, the tendon reflexes are lost and a reaction of degeneration develops.

The period of paralyzes lasts from one to six weeks, after which the movements are gradually restored.

The *restoration period* may last several weeks and even months. In some cases all affected muscles may be restored to normal; in other cases stable paralyses remain for life. These stable paralyses most frequently affect the deltoid and tibialis muscles. Most of muscles capable of functioning are restored to normal within six months. However, the movements may be restored only in the beginning of the second year.

Residual period. Eventually atrophy and grave deformities, sometimes even dislocations and subluxations develop in the affected muscles as a result of contractions of the antagonistic muscles. These affections cripple the child if no adequate orthopedic treatment is administered in the initial period of the disease.

Forms of the disease. The disease occurs in various forms which are distinguished by their severity and clinical manifestations.

The *typical form* is characterised by its sudden onset, the *preparalytic* stage lasting 3-4 days, the *stage of paralyses* usually more extensive in the beginning and selectively affecting most frequently the shoulder girdle, the deltoid and arm muscles and then the shank muscles. This stage lasts from six months to one year. Subsequently, stable paralyses of some initial affection, at times very negligible, remain. These *stable paralyses* are attended with muscular *atrophy* and, in cases of improper treatment, with *contractures* of the antagonists and *retarded growth of the extremities*. The brain is not affected and mental development is normal.

There are very many *atypical forms*. Some of them are manifested in so-called "effaced" forms in which some of the symptoms may be absent and all manifestations may be very mild, rendering the diagnosis of such cases very difficult; these forms may manifest themselves in sudden development of *paralysis of the facial nerve* and *temporary aphasia*—loss of the power of speech after a slight initial febrile period. In other cases of atypical forms the clinical picture, in addition to the usual symptoms, which are sometimes feebly marked, is characterised by meningeal symptoms so that the disease *simulates acute meningitis* (meningeal form). Some cases greatly involve the cerebral cortex (convulsive seizures, loss of consciousness); these cases represent the encephalitic form.

Differential diagnosis. The *preparalytic* stage of poliomyelitis may be confused with *croupous pneumonia* due to prolonged high fever and negative examination data.

Leukopenia, hidrosis and hyperesthesia are typical of this period of poliomyelitis. In the presence of catarrhs of the mucous membranes poliomyelitis may simulate influenza.

The *paralytic stage* may be mistaken for general *diphtheric* paralysis. In diphtheric paralysis there is angina in the anamnesis and various degrees of heart affection (myocarditis); lumbar

puncture reveals a considerable amount of protein (0.5-1%) which, however, is not the case during more advanced stages of poliomyelitis. Poliomyelitis is particularly easy to confuse with infectious (viral) polyradiculoneuritis. However, the onset of polyradiculoneuritis is slower, the temperature is not so high, and the patient has *sharp pains*. The paralyses in the spinal form of polyradiculoneuritis are peripheral, but the muscles are affected distally, i.e., the feet and wrists are affected most, while in poliomyelitis the affection of shoulder and thigh is more strongly pronounced. The cerebrospinal fluid shows elevated protein content and a negligible increase in cells. The prognosis of spinal forms of polyradiculoneuritis is complete recovery, unlike poliomyelitis which often leaves irreversible stable paralyses with atrophy.

Complications. *Bronchitis* and *pneumonia* are observed, especially in cases in which the intercostal muscles are involved or the *nuclei of the medulla oblongata* with the respiratory centres are affected. Nurslings suffer from diarrhea and otitis. Contractures and *deformities* develop as a result of the paralyses, stretched muscles and joints and resultant increased contraction of the unaffected antagonistic muscles.

Prognosis. The death rate is from 3 to 10 per cent, complete recovery—from 10 to 20 per cent, the remaining cases result in various deformities and stable defects. The younger the child the graver the sequelae of paralysis manifested in retarded growth of the extremities, locomotor disorders, and frequently complete atrophy of one or both extremities.

Treatment, care, hygiene. Early diagnosis and adequate treatment of the disease during its preparalytic stage are very important. Good results achieved by intramuscular or spinal administration of convalescent serum have been reported by several authors since 1915. The authors state that administration of the serum during the preparalytic stage prevents paralyses from further development or attenuates them.

The convalescent vaccine is obtained from persons who had poliomyelitis 2-3 months or even 1-3 years before.

Dosage: 10-20 ml into the spinal canal, 20-50 ml intramuscularly.

If it is difficult to obtain convalescent serum, the serum of healthy people in somewhat greater doses may be used.

The serum administered during incipient or advanced forms of paralysis stage, proves ineffective.

Methods of treatment during the paralytic stage. Proper care and hygiene are an important part of the treatment of the affected

child. In addition to a maximum of fresh air, bed rest, adequate nutrition, warm baths and, for nurslings, maternal milk, the child's correct position in bed and care of the paralysed extremities are also very important. The child must be kept in bed for at least two weeks after the acute symptoms have subsided. The mattress should be hard so that the body and legs are extended and the back is straight.

In paralysis of the foot the leg should be splinted; to prevent the paralysed foot from sagging it should be fixed in the normal position. In paralysis of the arm the arm is splinted. To avoid pressure upon the paralysed parts the blanket should be placed on to an arched grid.

During the acute period of the disease, especially in the presence of meningeal symptoms, the patients are treated with penicillin; intramuscular proserine (neostigmine) injections are now recommended.

Rp. Sol. Proserini hydrochlorici 0.05%,
DS. 0.5-1.0 intramuscularly once a day or
Rp. Dibasoli 0.002-0.003
DS. One powder a day per os

Infants under two years of age are given 0.3-0.5 ml and children past two years of age—0.5-1.0 ml of a 0.05 per cent proserine solution; dibazol is administered per os in the following doses: nurslings under one year—0.002 g, children from two to three years of age—0.003 g, from three to five—0.005 g, from five to eight—0.007 g, from eight to twelve—0.008 g. Treatment with proserine and dibazol is prescribed not earlier than 7-10 days after development of paralysis. Simultaneously, if there are no pains, massage and corrective exercise may be indicated. It is necessary to massage the muscles affected the most, since this improves the blood circulation in the paralysed extremities; exercise imparts strength to the intact muscles and prevents deformities in the affected limb. The massage and corrective exercise must be continued all through the first year and conducted with intervals during the subsequent 1-2 years of the disease.

Electrotherapy is recommended from the very days following the development of paralyse; the treatment consists in application of ultra-high frequency and transverse diathermy to the affected region of the spinal cord every other day (a total of 25-30 sessions). After an interval of 1-2 months the treatment may be repeated. About 4-6 weeks after the development of paralysis electrotherapy (faradisation and galvanisation) is administered to the affected muscles for a period of 1.5-2 months, the course

being repeated with 1-2 months intervals over a period of 1-2 years. Warm baths are salutary at all stages of the disease.

The *period of residual paralyses*, when various deformities not infrequently develop, requires orthopedic measures and surgical intervention; fangothrapy (mud baths) in Odessa, Yevpatoria, etc., and labour practice may be simultaneously indicated. Children with grave deformities require public aid and must be taught to work.

Prophylaxis. Measures should be taken to isolate the affected child as soon as possible. Patients may not be discharged from the hospital before 21 days from the onset of the disease. The general measures to be carried out, especially during epidemic flare-ups, include control of congestion, extensive aeration and treatment of nasopharyngeal catarrhs, the latter apparently facilitating viral infection of the mucous membranes.*

In the U.S.S.R. children are now being vaccinated against poliomyelitis en masse with the Sabin vaccine prepared from living but attenuated (non-neurotropic) strains of the poliomyelitis virus.

The vaccine is dispensed in pills, two pills being given to children at a time with an interval of three weeks after the first dose, and one month after the second and third doses. The total immunisation dose is eight pills.

Since poliomyelitis is contracted mainly through the gastrointestinal tract, the patients must be provided with separate dishes which should be sterilised by boiling; the patient's underwear and linens should be thoroughly disinfected. The room in which the affected child lived must be disinfected after his departure for hospital and the toys should be washed with boiling water.

EPIDEMIC CEREBROSPINAL MENINGITIS

Etiology. The pathogenic agent of epidemic cerebrospinal meningitis is the meningococcus. It is shaped like a coccus, resembling a coffee bean and usually arranged in twos or fours, and sometimes in clusters when viewed in a stained preparation made from a smear of the cerebrospinal fluid. It stains with the usual dyes, and is decolourised by the Gram method. Intracellular localisation of the microbes is characteristic of the smears taken from the cerebrospinal fluid and, especially, of the pus taken from the abscesses.

* On appearance of the disease in children's institutions the children are administered preventive inoculations with serum of adult persons (dose—

The meningococcus grows in media containing hemoglobin or ascitic fluid. Four types (A,B,C,D) are distinguished according to agglutination reactions.

The meningococcus excretes a toxin; it may be present in the patient's cerebrospinal fluid, pus (in joint affections) and blood. Not infrequently it is found in the nasopharyngeal mucus.

Epidemic cerebrospinal meningitis occurs mainly in children under 14 years of age (80 per cent) and predominates in early childhood (up to 40-50 per cent of nurslings as against the other children's age-groups).

The youngest children (up to three months) are affected less frequently due to the immunity acquired from their mothers through the placenta and the mother's milk. But even the children of this age-group (the newborn included) may contract the disease already during the very first weeks of life. *Poor living conditions*—filth, lack of fresh air, malnutrition and, especially, *congestion* all play an important part in spreading the disease. Various nasopharyngeal catarrhs are predisposing factors for this disease.

The disease is transmitted to children from adult bacilli carriers who are not ill or whose illness is not manifested in meningeal inflammation, but in some vague febrile affection marked by symptoms of influenza or angina during an epidemic flare-up.

Pathogenesis. According to the modern concept, epidemic cerebrospinal meningitis is a *general hematogenous infection*. The microbe enters the organism through the nasopharynx and upper respiratory tract and quickly penetrates into the blood, from which a culture can in some cases be made; it soon disappears from the blood which has high bactericidal properties and localises itself in definite tissues and organs; the meninges are affected most frequently and rapidly, for which reason the whole process sometimes seems like an isolated affection of the meninges. In some cases, however, the meningococcus infection may have other localisations (choroid, joints, skin) where the process is manifested in the symptoms of general intoxication and general meningococcal affection (meningococcemia).

Pathologic anatomy. In acute cases of epidemic cerebrospinal meningitis the pathoanatomical changes are limited to acute purulent inflammation of the pia and arachnoid of the brain and spinal cord. Not infrequently substance of the brain and spinal cord is also involved. The cerebral vessels, especially the veins of the base of the brain, are often inflamed (vasculitis) and become obliterated as a result of the inflammation; the process may extend to the *ventricles of the brain*, affecting the ependyma (ependymatitis) and vascular plexuses of the ventricles (choroiditis) which usually lead to death.

In cases of full recovery all inflammatory phenomena may clear up completely, in other—graver—cases the inflammation may cause thickening of the meninges, adhesions, defects in the cortex and destruction of the nerves, which may lead to a number of grave sequelae and complications (hydrocephaly, paralyses, deafness, blindness, ankylosis). Complications caused by the vulgar flora (diplostreptococci), i.e., otitis, gastrointestinal disorders, (diarrhea)

and pneumonia are not infrequently observed in children, especially in very young children. In cases of meningococchemia the septic processes in various organs are associated with meningococcal embolism (skin, joint, choroid).

Clinical findings. The incubation period is from two to five days. The onset of the disease is usually acute and rapid. The temperature suddenly rises to 40° and higher, vomiting and, in severe cases, convulsive seizures appear; older children complain of headaches. In nurslings marked unrest, refusal to take the breast and sometimes rather early protrusion of the fontanel due to increased formation of cerebrospinal fluid, are observed. Hyperesthesia and pain in the muscles are also noted.

Meningeal symptoms—occipital rigidity, changes in the muscle tone of the extremities, Kernig's and Brudzinski's signs, red der-

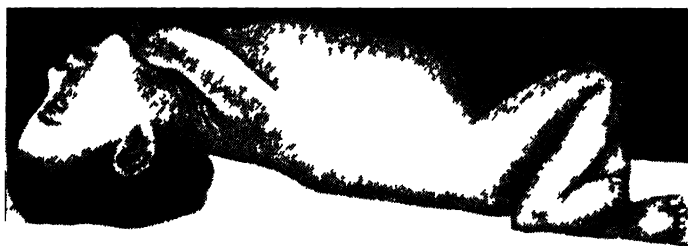


Fig 38 Position of the child suffering from epidemic cerebrospinal meningitis, his head thrown backwards

mography—develop very rapidly. In severe cases the child lies on one side with his head thrown back and legs flexed in the knees and pressed against the abdomen (Fig. 38). Examinations of other organs do not reveal any significant changes. The skin is dry, sometimes herpetic. The lungs and heart are normal, the heart sounds—clear; the pulse is considerably accelerated; the blood pressure is normal or slightly increased. The liver and spleen are not enlarged. The intestines function normally or show a tendency to constipation. Only the nurslings sometimes have liquid stool. The pharynx is usually unaffected; the tongue is but slightly coated. In very young children—moderate pharyngeal catarrhs as well as catarrhs of the upper respiratory tracts are observed.

The consciousness is usually clear. The appetite is moderately disturbed. Lumbar puncture reveals the cerebrospinal fluid to be turbid with purulent sediment containing neutrophils (when allowed to stand); the general protein is increased and Pandy's reaction is positive; meningococci are found in the smears.

- Subsequently the process may run now a milder and now a graver course, depending upon the *intensity of general phenomena of intoxication* (the action of toxins, affecting central nervous system and cardiovascular apparatus), the *gravity of meningeal affection* and the presence and severity of complications which in turn depend mainly upon the time the treatment was started.

Diagnosis. In typical cases marked by acute onset and rapid development of meningeal symptoms epidemic cerebrospinal meningitis may be confused with croupous pneumonia, relapsing fever and scarlet fever, which at first not infrequently exhibit meningeal symptoms. Pulmonary affection which is a symptom of pneumonia, angina or developing necrosis and eruption which attend scarlet fever, as well as an enlarged spleen which indicates relapsing fever, must not be overlooked. Purulent nonspecific (diplostreptococcal) meningitis, bacilligenic influenzas, meningitis and acute meningoencephalitis may be characterised by a similarly rapid onset. The onset and course of tuberculous meningitis are much slower. Mumps may also exhibit a meningeal syndrome.

The decisive date for diagnosing the disease is furnished by a spinal puncture.

Normal cerebrospinal fluid is transparent, does not form a film when allowed to stand, and its sediment contains 2-3-4 formed elements in the field of vision.

In epidemic cerebrospinal meningitis the fluid becomes turbid or even purulent. When allowed to stand it forms a leukocyte sediment and not infrequently a thick film. The number of cells is increased, sometimes very sharply.

Examination of smears reveals a prevalence of neutrophils with meningococci both intra- and extracellularly; the meningococci are grown in special media and are agglutinated to determine the type.

In purulent meningitis of other etiology the fluid is turbid or purulent; smears show neutrophils and the microbes causing the inflammation (Gram-positive streptococci, Gram-positive diplococci and Gram-negative Hemophilus influenzae).

In tuberculous meningitis the fluid is transparent and forms a film when allowed to stand; stained by the Ziehl-Neelsen and fixed on a slide it shows tubercle bacilli.

Course of epidemic cerebrospinal meningitis of moderate gravity. The temperature produces a peculiar, gradually descending curve consisting of a number of waves fluctuating between 37 and 39.5°. The symptoms disappear gradually. In acute cases the fever period lasts about 3-4 weeks; the break in the protein curve of the cerebrospinal fluid and other indices (blood, weight, etc.) occurs much sooner, approximately during the first 10-20-25 days. In protracted cases fever and all the other symptoms may persist for several weeks and even months, the break in the protein curve occur-

ing later (one and a half months and still later) or the protein curve running a fluctuating course.

Milder cases are characterised by a shorter course, feebly marked meningeal symptoms, lesser intoxication, fewer and milder complications.

In *more severe* cases all these symptoms are more strongly pronounced.

Formerly epidemic cerebrospinal meningitis ran a particularly grave course in infants and was responsible for 50 per cent and more of their mortality; not infrequently the disease was followed



Fig. 39. Marked hydrocephaly in five-month-old child associated with severe form of epidemic meningitis

by severe complications: paralysis, hydrocephaly (Fig. 39), ependymitis (inflammation of the lining membrane of the ventricles of the brain and the adjacent cerebral tissue) (Fig. 40).

Of the surviving children only a few remained entirely healthy, while most of them retained grave lifelong defects of the central nervous system (paralyses, partial or complete blindness or deaf-mutism, hydrocephaly and retarded mental development).

Now that meningitis is treated with such effective agents as sulfidine (sulfapyridine), sulfazole (sulfamethylthiazole) and other sulfa drugs, as well as penicillin, the clinical course of the disease has considerably altered, grave complications are almost absent and mortality has sharply decreased.

In some cases of epidemic cerebrospinal meningitis symptoms of general meningococcal infection of the organism are observed in addition to the meningeal symptoms; in addition to the usual general symptoms (pyrexia, headaches, inappetence, etc.) the meningococcal infection is sometimes characterised by cutaneous

affections, such as various eruptions, especially the characteristic stellate hemorrhagic eruption with subsequent necrosis in the centre, joint affections in the form of swollen finger, toe, ankle and wrist joints sometimes with a purulent exudate in the joints (Figs 41 and 42).

In rarer cases, in addition to meningeal affections, there is also an inflammation of the uvea (uveitis).

The meningococcic nature of this type of affections is established by the presence of meningococci in every eruption, in the blood, and in the purulent exudate of the affected joints.



Fig 40 Characteristic position of extremities of eleven-month-old child affected with epidemic meningitis complicated with endymenitis

If all these affections are evident, the patient is said to have *meningococcemia*. Clinical observations have shown that in many affected nurslings this meningococcemia usually precedes the involvement of the meninges and is therefore a kind of prodrome of meningitis.

Not infrequently careful questioning of the mother reveals that at the time of the sudden onset of the disease, characterised by a sharp rise in temperature, the child had an eruption (mainly on the extremities and less pronounced on the body) resembling insects' bites; in some cases this eruption persisted for several hours (up to 24) and in others for a longer time when the meningeal symptoms were already evident.

There were also cases in which meningococcemia, already in progress, remained all through the illness with no meningeal symptoms, cerebrospinal fluid being normal.

In such cases the child is said to have meningococcemia without meningitis, or pure meningococcemia.

The outcome of such affections, if treated adequately and in due time, is always favourable, unless the meningococcemia is hypertoxic from the very onset, i.e., a kind of grave, fulminant septicemia ending lethally during the first 24 hours. But such cases are extraordinarily rare.

Prognosis and mortality. Epidemic cerebrospinal meningitis is a

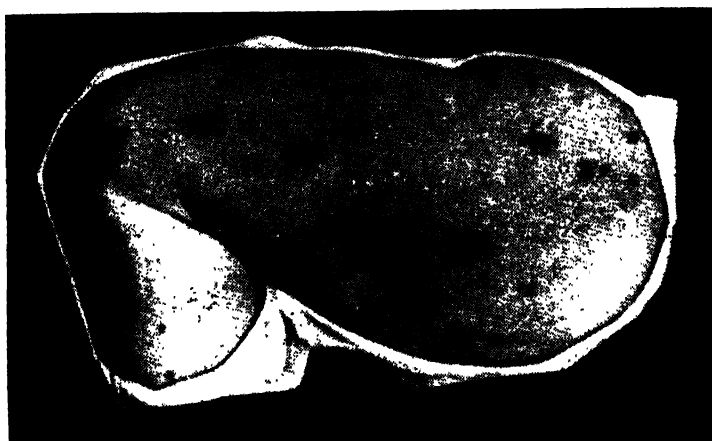


Fig. 41. Stellate hemorrhagic eruption in meningococcemia

grave and dangerous disease, especially for very young children. However, if treatment is started in due time, i.e., during the first hours or days, it ends favourably in the overwhelming majority of cases and leaves no mental or physical defects. But in cases of late treatment the patient may retain the afore-described residual phenomena which lead to invalidity if he does not die soon, as was formerly observed in grave, especially hypertoxic, cases.

Hygiene, care, treatment. Proper care, hygiene and diet are very important factors in the treatment of this disease.

The patient's room must be thoroughly aired by keeping the windows open for long periods of time. In summer the windows of the patient's room may be open day and night or the patient may be kept outdoors. Hot-water bottles must be used to keep the patient warm during the cold time of the year. There must be enough light in the room, which may be temporarily darkened (curtains, screen) only when the patient shows signs of great cortical excitation, convulsions, affections of the eyes and photophobia.

The *skin* is often affected in meningitis (eruptions, hemorrhages with subsequent ulcers); in grave forms the patient easily develops bedsores, for which reason he must be made to *turn from side to side* and a *rubber ring must be placed under him*. The linens must be frequently changed. In cases aggravated with deep ulcers and bedsores sterile gauze with sterile vaseline must be applied to the affected parts of the skin.

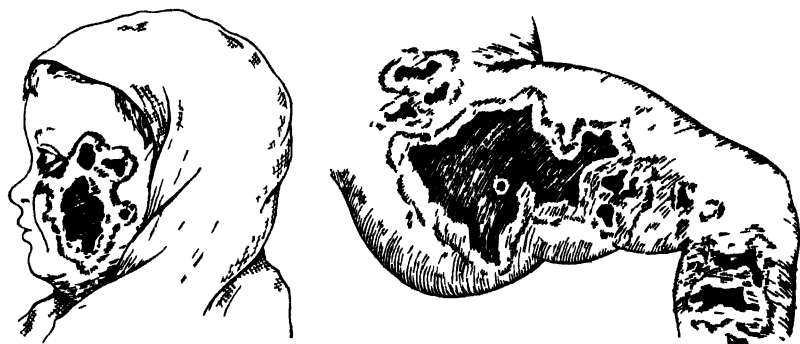


Fig. 42. Severe cutaneous lesions in meningococcemia. Hemorrhages with subsequent skin necroses in seven-month-old girl

Hydrotherapy is the most important agent in controlling the symptoms of intoxication and the affections of the central nervous system; in cases of fever and *headaches* application of *ice* or cold, frequently changed lotions to the head are indicated.

Baths are one of the important agents.

Hot baths (38-40°C) for ten minutes considerably reduce the general muscular tension and relieve headaches. In grave cases baths may be taken twice a day. Their initial temperature of 37°C is boosted in the course of 3-5 minutes to 40°C, the patient staying in the bath for another 5-8 minutes. Cold compresses are applied to the head.

The *diet* being very essential, the most serious attention should be devoted to the patient's proper diet.

Breast-feeding is very important for *nurslings*. Artificial-fed infants should be given human milk. It is necessary to give the patient enough *vitamins* (juices, fruit, fresh vegetables, cod-liver oil, egg yolk, kefir).

Medication. As soon as epidemic cerebrospinal meningitis has been diagnosed the child should be given one of the sulfa drugs, preferably sulfidine, sulfazole or sulfadiazine. For the first 2-3 days

the dose should be 0.3 g per 1 kg of the child's body weight a day in 3-4 intakes; during subsequent days the dose should be increased to 0.2 per 1 kg of the child's weight, the course of treatment lasting for 10-14 days. Treatment with sulfa drugs should be prolonged to three weeks in case pyrexia and changes in the cerebrospinal fluid persist. Simultaneously penicillin is administered in a daily dose of at least 10,000 u per 1 kilogram of the child's weight. Of this amount not more than 25,000-30,000 u is injected into the spinal canal, the remaining dose being divided into two equal parts and administered intramuscularly. For the first three days penicillin is injected into the spinal canal every day, 10-20 ml of the cerebrospinal fluid being removed beforehand; after that penicillin is administered every other day. A total of 6-7 injections should be made into the spinal canal. The concentration of penicillin to be injected into the spinal canal is 25,000 u per 1 ml of physiologic saline solution. In some cases administration of penicillin into the spinal canal produces side effects, namely, convulsions, temporarily clouded consciousness and cardiac weakness; to prevent these phenomena penicillin should be administered slowly with the patient lying in bed without pillows; in case of considerable convulsions the patient should be given a chloral hydrate enema and hot-water bottles should be applied to his feet; camphor and cordiamine (nickethamine) should be administered in cases of cardiac weakness. As a rule, these symptoms do not persist for long and are not dangerous. The following symptomatic drugs are used in the treatment of the disease: in a state of excitation—a 2 per cent solution of bromides, for convulsions—a 2 per cent chloral hydrate solution in an enema (50 ml per enema for older children and 25-30 ml for younger children, 2-4 times a day).

For relieving *headaches*, systematic (two times in five days) lumbar punctures during which 10-20-30 ml of fluid are removed may possibly influence the course of the inflammatory process.

In case of developing *uveitis* with pain in the eyes, cold compresses and later moist warm compresses are applied to the affected eye; 0.5 per cent atropine with cocaine is instilled into the conjunctival sac to rupture it and prevent the formation of synechia by way of maximal dilation of the pupil.

In *joint affections* splint and warm compresses are applied to the affected joint; in cases of considerable accumulation of pus the latter is aspirated with a syringe. No incisions should be attempted too soon, for the pus is not infrequently reabsorbed spontaneously unlike other forms of suppurative arthritis. Small incisions may be required only in cases of large accumulations of pus.

In paralysis of the extremities—splint to maintain a correct position, and later massage.

In paralysis of the urinary bladder—a bedpan should be placed under the patient, emptying of the bladder being watched all the time; in cases of urinary retention—hot baths, hot-water bottles to the abdomen, careful expression of the urine by pressure applied to the abdominal wall, in extreme cases—catheterisation.

Chafing must be prevented.

In cases of hydrocephaly—systematic withdrawal of the fluid (with adhesions present in the canal the fluid is removed by *cisternal puncture*, in nurslings—by *fontanel puncture*); in severe cases 50-100 ml of fluid may have to be withdrawn.

Such complications as otitis, pneumonia and intestinal disorders are treated as usual.

Prophylaxis. The child should be taken to the hospital; he may be left at home only if necessary precautions are taken to prevent the spread of the infection.

Current and final *disinfections* should be made, the patient *isolated* and the people surrounding him *quarantined*.

Since the disease may be spread through meningococci carriers, those who have had contact with the patient should gargle the throat, observe the rules of hygiene and spend a good deal of time outdoors; they should not be allowed to come in contact with nurslings. Mucous catarrhs of such persons should be treated.

The population must be taught to stay as long as possible outdoors, to keep their homes clean and avoid congestion.

No specific vaccination has been elaborated as yet.

ENDOCRINE DISORDERS, DISEASES OF METABOLISM AND GROWTH

The glands of internal secretion—endocrine glands—have no ducts and secrete their products—*hormones*—directly into the blood stream. The hormones influence growth, metabolism, nutrition and the functioning of the nervous system. The central nervous system, the cerebral cortex in particular, in its turn influences the endocrine glands and regulates their functions.

The endocrine glands include the *thyroid*, *parathyroids*, *thymus*, *hypophysis*, *pineal body*, *adrenals*, and partly the *pancreas*, namely the islands of Langerhans.

All the endocrine glands are closely interdependent, and a disturbance in the function of one causes disorders of the functions of the others. Sometimes, however, the inadequate activity of one gland stimulates intense and additional functioning of the others, and, owing to such intimate connections, the organism maintains the requisite hormonal balance. Often no such equilibration takes place and the result is a complex of morbid symptoms produced by the improper functioning of a number of endocrine glands. This

is why it is often hard to tell the affection of which gland and what particular disturbance have influenced the development of the clinical picture of the disease and for this reason a good deal in the pathology of endocrine diseases still remains obscure.

However, several diseases of the children's endocrine-vegetative apparatus are rather well known and in these cases it is possible to indicate which of the particular endocrine glands are affected. Some of these affections develop during the intrauterine period, the disease begins early in life and is of the nature of a *congenital disease*. In other cases the onset of the disease is associated with some general affection—infection, development of a tumour or gumma in a gland or adjacent area, or cerebral disease which may be followed by endocrine disorders; affections of the vegetative nervous system are also in large measure conducive to the onset of endocrine disorders.

Diseases of the endocrine glands are responsible for about 1.9 per cent of child mortality.

ADIPOSOGENITAL DYSTROPHY

Adiposogenital dystrophy is an endocrine disease whose most prominent symptoms are excessive adiposity of the child and underdevelopment of the child's external genitalia (Fig. 43).



Fig. 43. Adiposogenital dystrophy in eleven-month-old child

The disease is based on a disturbance in the functions of the posterior lobe of the *hypophysis* sometimes due to organic changes in the gland (tumours, gummas of the hypophysis or adjacent cerebral areas). Sometimes the child begins to develop such adiposity as a result of an inflammation of the meninges, connected with development of hydrocephaly in the region of the third and fourth ventricles.

Clinically the disease is characterised by considerable adiposity, the adipose tissue being deposited mainly in the regions of the mammary glands, the abdomen, buttocks and the mons pubis. The symptoms always include under-

development of the external genitalia and retarded sexual development (late menses, failure of the boy's voice to change).

No mental changes are observed in cases of adiposogenital dystrophy except instances in which the dystrophy is combined with other endocrine disorders (thyroid insufficiency).

Prognosis. This disease in itself is not dangerous if there are no organic lesions in the region of the hypophysis (tumours, gumma).

The *treatment* depends in each individual case on the nature of the disease: suspicion of syphilis warrants specific treatment; in cases of clear symptoms of tumours surgical intervention is sometimes resorted to. All other cases indicate dietetic treatment which is generally administered in all instances of adiposity, the diet consisting of vegetables, fruit, little bread, meat and fats with sweets and pastry completely excluded.

This disease has of late been treated with endocrine preparations (hypophyséal, thyroid, gonadal). Combined treatment (hypophysin, thyroïdin, ovarin) is usually prescribed. This treatment is best conducted in clinics or hospitals since it requires strict control of the patients' metabolism.

DIABETES MELLITUS*

This disease is characterised by a continuous discharge of sugar with the urine and rather extensive nutritional disorders.

The disease affects children less frequently than it does adults, and particularly rarely very young children. It occurs more often in children past 6-7 years of age.

Etiology. The disease is due to a disorder of the function of the pancreas, particularly its islands of Langerhans which are of an endocrine nature and secrete a special hormone—insulin—required by the organism for normal carbohydrate metabolism.

Sections of these islands taken from persons who died of diabetes often show these islands to be modified. The causes of such modification are not yet sufficiently clear, infection possibly constituting a contributory factor.

An important part is played by disorders of the central and vegetative nervous systems whose functioning is intimately connected with that of the endocrine glands, the functioning of the pancreas in particular.

Clinical findings. In the beginning the disease in children often fails to be noticed; diabetes is usually suggested by the appearance of intense *thirst and the passage of a large quantity of urine*; the final diagnosis is made on the basis of a urinalysis when sugar is discovered in each portion of the urine, often in continuously in-

* The term comes from the Greek and means a discharge of sweet.

creasing amounts. A urinalysis is sometimes occasioned by the appearance of the odour of acetone exhaled by the child.

In addition to the intense thirst and discharge of large quantities of urine the child becomes generally weak and emaciated despite his good appetite; sometimes the process is characterised by excessive hunger. A constantly dry skin is observed.

Subsequently the patient easily tires and in the end may develop a comatose condition (*diabetic coma*)—unconsciousness, unrest, odour of acetone from the mouth, and convulsions during which the child may die with symptoms of cardiac failure. The coma is due to poisoning of the organism with ketone bodies—products of disturbed lipid and fat metabolism.

The diagnosis of diabetes is decided by an analysis of the urine for sugar, although in nurslings not all forms of glycosuria (the presence of sugar in the urine) will necessarily be of diabetic origin; in acute digestive disorders and in certain infections sugar is not infrequently found in their urine, the sugar disappearing during their convalescence. In a number of diseases sugar is very seldom found in the urine of older children, and for this reason, if prolonged glycosuria is observed during or after infections, the possibility of diabetes must always be considered.

In diabetes the prognosis is serious because the child may die of coma; nutritional failure and, often, acute emaciation predispose to all manner of infection which is poorly tolerated by the patients; infection considerably aggravates the course of diabetes and not infrequently leads to death. Only timely treatment and administration of insulin are likely to save the child; the course of diabetes in adults is much more favourable.

Treatment. Soviet scientists have proposed a new method of treating diabetics, namely, prescription of a normal diet which meets the physiological requirements of the organism and contains the necessary proportions of proteins, fats and carbohydrates, with simultaneous administration of such quantities of insulin as may ensure the most complete assimilation of the carbohydrates consumed with the food.

Before the discovery of insulin* the treatment of diabetes boiled down mainly to dietotherapy; the diabetics were kept for a long

* Insulin is an alcoholic extract from the pancreas of cattle. It is dosed in units of its action on the organism, in the sense of its reduction of sugar in the blood, rather than in grams. The laboratories producing insulin regard as a unit of its action the amount which suffices to reduce in four hours the sugar in the blood of a 2 kg rabbit, starved for 24 hours to half its initial contents. The clinical insulin unit equals one-third the rabbit unit. The method of its production was forecast by L. Sobolev, a Russian scientist, as early as 1901.

time on a low carbohydrate diet. In childhood, however, a prolonged strict diet usually leads to grave nutritional disorders. For its growth and development a child's organism requires an adequate diet containing enough proteins, fats and carbohydrates; failure to provide enough of these constituents was responsible for the high mortality from diabetes among children, especially among very young children.

The discovery of insulin has made it possible to prescribe an adequate and sufficiently calorific diet for the treatment of diabetes.

If diabetic children are now in any way limited in the consumption of carbohydrates, it is done only for the first few days of the disease to determine their tolerance for carbohydrates and, depending on that, to prescribe a definite dose of insulin which is individual in each particular case. The therapeutic dose of insulin is established on the basis of 1 u per 5-6 g of sugar in the daily urine.

If a child is admitted to a hospital in a comatose state, subcutaneous injections of insulin are prescribed at once, the preparation being administered in a dose of 20-30 u twice at a one-hour interval; subsequently the insulin injections are repeated every two hours in a dose of 10-20 u until disappearance of the acute symptoms of diabetic coma (return of consciousness, normal respiration). Simultaneously with the insulin injections the patient is given to drink a 5 per cent glucose solution or is administered glucose per rectum (100-300 ml of a 5 per cent solution). In grave cases of coma glucose (20-40 per cent) has to be administered intravenously, but never intramuscularly or subcutaneously because hypertonic glucose solutions easily produce necrosis of the skin.

It should be remembered that administration of large doses of insulin with a sharp limitation of carbohydrates in the child's diet, as well as the insufficiency of glucose given during diabetic coma, may cause a considerable drop in the sugar level of the patient's blood (hypoglycemia) which will manifest itself clinically in the form of *hypoglycemic coma* characterised by vomiting, bradycardia, headache, occipital rigidity and sometimes convulsions. In such cases it is necessary to administer enough sugar per os (30-40 g) or 300-400 ml of a 5 per cent glucose solution; at this time insulin is contraindicated.

In addition to the aforesaid treatment it is necessary to observe the general rules of hygiene, including baths and keeping the child outdoors; in grave cases the patients must be confined to bed.

The course of diabetes may be aggravated by any disease, for which reason after discharge from the hospital the diabetic child must be kept under constant observation by the district physician,

the treatment with insulin being conducted under his guidance; in cases of aggravation the child must be hospitalised.

Table 16

Differential Diagnosis of Diabetic and Hypoglycemic Coma

Indices	Diabetic coma	Hypoglycemic coma
Character of respiration	Deep, without pauses	Normal alternation of inhalations and exhalations
Pulse	Small, quick	Slow (bradycardia)
Condition of the skin	Dry	Sweaty
State of the nervous system	Unconsciousness, without convulsions	Occipital rigidity, convulsions, vomiting
Sugar content in the blood and urine	Hyperglycemia Glycosuria	Normal or hypoglycemia (no sugar in the urine)

It must also be remembered that diabetes often causes complications of the skin (pyoderma, furunculosis), stomatitis, affections of the kidneys and aggravation of tuberculosis.

DIABETES INSIPIDUS

This disease characterised by intense thirst seldom occurs in children; affected children consume large quantities of liquid and correspondingly excrete extensive amounts of urine which contains no sugar.

The *etiology* of the disease is connected with functional insufficiency of the posterior lobe of the hypophysis which secretes an *antidiuretic hormone* (diminishing water excretion); this insufficiency is characterised by retarded mental and physical development.

The *etiology* of the disease is as yet insufficiently clear, although it is supposed that the disease is largely caused by a combined disturbance in the functions of several endocrine glands (pluriglandular disorder). Symptoms of thyroid insufficiency are not infrequently observed, the functioning of the thyroid being disturbed during the extrauterine period; the cause of this disturbance is not clear, although illnesses of the mother and intoxication of the fetus may have something to do with it.

Autopsy reveals insufficient development of separate lobes of the brain and late development of the cerebral cortex; the convolutions are of an embryonic nature.

In some cases of diabetes insipidus sections show tumours of the posterior lobe of the hypophysis.

Clinical findings. The picture of the disease is characterised by intense thirst; the child loses his appetite for solid food and develops a constant craving for water and other liquids. Children affected with this disease lose weight, their skin becomes dry, perspiration and salivation diminish. In some cases retardation of physical development is observed.

Sharp limitation of liquid consumption gives rise to headaches, vomiting and excitement. Analysis of such children's urine shows nothing pathological except its low specific gravity.

Diabetes insipidus must not be confused with the habit of consuming a lot of liquid observed in some children; in such cases the amount of liquid consumed will usually not be so large and the thirst will disappear as soon as certain educational measures are employed.

Diabetes insipidus is treated with injections of hypophyseal preparations. These preparations considerably diminish thirst, but their action is also temporary and it is usually difficult to effect a permanent cure. The daily dose is 0.5-1.0. The most effective treatment today is administration of adiurecrin—a preparation of the posterior hypophysis—into the nose. The dose is 0.01 three times a day.

MYXEDEMA

This is a disease which, in addition to affecting the child's mentality, is characterised by considerable changes in his appearance, mainly in the face, due to development of a peculiar edema, hence, the name of the disease meaning mucous edema; it is also characterised by considerably retarded growth (Fig. 44).

The disease is congenital and is due to altered functioning of the thyroid gland, most frequently involving its total aplasia.

Clinical findings. Soon after birth the clinical picture of the disease is as yet insufficiently clear, and it is sometimes difficult to diagnose the case since, during the fetal stage, the lacking hormones of the atrophied gland are supplied by the maternal circulatory system and after birth the child receives them with his mother's milk. Only as the child grows older, when the lack of this hormone becomes increasingly more manifest, espe-

cially after weaning, do the symptoms of this disease come to the fore.

The picture of the disease is particularly characterised by a change in the child's appearance: a stupid expression, flat saddle-shaped nose, thick lips, mouth always half open with a large protruding, and as though swollen, tongue. The hair on the head is usually hard and short, the skin is dry and has a yellowish tint. The skin on the neck appears thick, as though edematous, although there is no real edema since pressure with a finger produces no in-



Fig 44 Myxedema in two-year-old child

dentation. The limbs are thick and terminate in clumsy, short fingers and toes; the child's movements are sluggish, delayed ossification and retarded growth in length are observed; the patient looks like a dwarf and has a gruff voice. In addition, there is a delayed development of the genitalia and greatly retarded sexual maturation. The mental development is deeply impaired

In the absence of requisite treatment the prognosis is very unfavourable.

Treatment. The treatment consists in administration of thyroid preparations. Regular administration three times a day of 0.01 of *thyroidin* for every year of life must be instituted in earliest childhood, as soon as the first signs of the disease are observed. The maximum daily dose for older children is 0.06-0.1; it is rarely increased to 0.2.

Rapid improvement is usually observed during the very first 4-6 weeks of such treatment; the treatment has to be continued all through life since only this warrants the hope that the patient will

be able-bodied, will not need constant aid and will not have to be committed to a special institution.

The later the treatment is begun, the less chance for success; but even in such cases the treatment must not be relinquished for it will to some extent improve the patients' mental state and the patients may thus be taught to do some, even if simple, work.

THYROTOXICOSIS

Increased functional activity of the thyroid gland produces a number of characteristic changes in the organism, which yield a peculiar picture of the disease long known as *Basedow's disease*.* Today this disease is designated by the term thyrotoxicosis.

In children thyrotoxicosis occurs very rarely and is most frequently observed in girls during the initial period of sexual maturation. At this age the thyroid gland is not infrequently enlarged, although there are no other morbid symptoms characteristic of Basedow's disease.

In a number of cases the causes of thyrotoxicosis cannot be determined. Sometimes the disease is observed to have been influenced by a severe nervous shock (fear); some authors believe the affection to be associated with past infectious diseases; however, it is difficult to determine the etiological factor in each individual case.

Clinical findings. In children the picture of this disease consists of the following characteristic symptoms:

Cardiovascular symptoms. The initial symptom is well-defined *tachycardia*; the heart is not infrequently hypertrophied or dilated. In some cases a nonorganic murmur is auscultated and pulsation of the cervical vessels is observed.

Eye symptoms. Exophthalmos combined with tachycardia is often an initial symptom of the disease; characteristic of thyrotoxicosis is the lagging of the upper eyelid when the patient slowly looks down, a white strip of the sclera remaining visible above the iris (von Graefe's sign).

Enlargement of the thyroid gland in the form of a goitre. Although this enlargement is not so pronounced as it is in adults, it is also one of the manifestations of thyrotoxicosis in children.

The symptoms include moist skin, slightly elevated temperature, a tendency to frequent defecation, emaciation, intense thirst, a

* Named after Karl Adolph von Basedow, the physician who was the first to describe this disease in 1840.

sense of fear, anxiety and excitement, increased metabolism and tremor.

Thyrotoxicosis runs a milder course in children than it does in adults, but sometimes the disease assumes a severe form which jeopardises the child's life.

Death may be caused by cachexia with intercurrent pneumonia, and aggravation of tuberculosis.

Treatment. In children, as in adults, the disease is treated with microdoses of iodine.

Iodine is prescribed either in the form of a 0.25 per cent potassium iodide solution (half a teaspoonful a day) or in the form of pills:

Rp. Jodi puri 0.01
Kalii jodati 0.1
Extr. Valerianae 2.0
Massae pilul. q. s. ut fiat pil. No. 40
DS. 1 pill once or twice a day after meals

The pills are administered for 20 days after which their administration is suspended for 20-30 days and then resumed for the same period of time. The course of treatment is 4-6 months.

Children with a severe form of thyrotoxicosis must be ensured maximum tranquillity and a nourishing diet.

In severe cases of thyrotoxicosis, in which this treatment proves ineffective, surgical intervention is indicated (excision of a considerable part of the thyroid gland).

MONGOLIAN IDIOCY

In 1866 Down described a peculiar congenital disease. The following symptoms of this disease strike the eye: a brachycephalic type of head* with a flat face and slanting eyes with epicanthus (a vertical falcate fold of skin covering the inner canthus); a slight protrusion of the eyeball and strabismus, and not infrequently rubicund cheeks (the child looks like a clown) are observed. Moreover, the patients have short fingers and toes, the fifth fingers and toes being somewhat flexed because of the shortened middle phalanges; they also have deformed ears. Auscultation of the heart not infrequently reveals a murmur, further examination discovering congenital heart disease. The patients also exhibit muscular hypotonia and weakness of articular ligaments, owing to which they perform extensive movements with the arms and legs.

Usually these children are very active, merry and inclined to imitation and grimacing. They are considerably underdeveloped mentally and sometimes exhibit a progressive development of idiocy.

* Brachycephaly—shortness of the head as distinct from dolichocephaly—the condition in which the head is much longer than it is broad.

Morbidity among such children is very high and most of them die of various diseases at a very early age, no more than 25 per cent surviving to adulthood.

Some authors recommend treatment of Mongolian idiocy with thyroidin, but the effect of this treatment is very weak.

Treatment with thyroidin is indicated more in cases of thyroid insufficiency.

CHONDRODYSTROPHY

Chondrodystrophy or achondroplasia is a congenital disease characterised by retarded growth, the retardation being due to a delayed growth of the limbs, while the trunk develops normally.

The etiology of this disease is not yet entirely clear. A certain influence may be exerted by infection or intoxication on the part of the mother, owing to which the cartilage cells located on the border of bone growth undergo certain changes during the fetal stage. Only the bones developing from cartilage are affected (the bones developing from connective tissue are not affected); hence, the peculiar defect in which some bones grow normally (the trunk, vault of the skull), while other bones (limbs, face and base of the skull) lag in their development, the bones growing in width rather than in length since the periosteum grows normally. In the most clearly defined cases this deformity is noticeable at the very birth; more often it becomes apparent several months after birth, the retardation in growth subsequently becoming more and more obvious. The following features strike the eye and characterise the appearance of chondrodystrophics: the arms are too short, the hands failing to reach to the middle of the thighs, as is the case normally, and barely reaching to the navel; short legs with a normal trunk, a large head with a small face, due to delayed growth of the facial bones and the base of the skull, and a normal vault of the skull. Such dwarfs produce the impression of well-developed and brawny people. The mental development of such people is normal and at a mature age they often attract the attention of those around them by their wit; in olden times chondrodystrophic dwarfs usually served as jesters in kings' courts.

There is no treatment; attempts to treat this disease with thyroidin and hypophyseal preparations at an early age prove futile.

ACUTE INFECTIOUS DISEASES

METHODS OF STUDYING ACUTE INFECTIOUS DISEASES

The theory of infection has gone through several stages of development.

The great achievements made by bacteriology during the second half of the 19th century and connected with the discovery of microbes—the causative agents of infectious diseases—furnished a basis for the further development of the theory of infection and for a long time determined the microbiological trend in studying infection.

This trend dominated until very recently and found its reflection in the views concerning a number of fundamental questions in the theory of infection: etiology and pathogenesis, peculiarities in the course of acute infections, the organisation of their therapy and prophylaxis.

The leading role in an infectious disease was believed to be played by its causative agent, the aggregate of whose pathogenic properties (i.e., virulence) determined the initial gravity of the disease, as well as its subsequent course and outcome. The state of the organism and its environment was either given little consideration or was completely disregarded.

It was believed that an infectious disease owed its onset to the direct influence of the pathogenic principle on the cells of the organism, while its development was due to the spread of this influence of ever greater cellular areas; it was held that the clinical and pathoanatomical picture of a disease was essentially determined by the specific ability of the causative agent selectively to affect definite tissues and organs and produce changes in them.

This localistic approach did not reflect the whole essence of infectious disease with its influence on the integral organism and did not take into account the nervous (reflex) mechanisms in the onset and development of disease.

The pathogenesis of the infectious process was interpreted on the basis of recognising two aspects of microbial influence—toxic (conditioned by toxins) and septic (connected with the microbial invasion of the blood and organs). The therapy of infections was accordingly built on the principle of fighting the microbe and its toxins and was elaborated mainly in this direction (antitoxic and antimicrobial serums and vaccines).

Such were briefly the views regarding some of the fundamental questions of the theory of infectious diseases, which prevailed during the first stage of their investigation.

Despite the significance and value of a number of observations and data furnished by the investigations conducted during that stage, the one-sided microbiological trend was unable to ensure further development of the theory of infection. Without due consideration of the importance of the macroorganism and the state of its reactivity many clinical facts remained unexplained, for example, the differences in the gravity of the course of infectious diseases with the selfsame source of their origin, the cyclic nature and fluctuations of the clinical manifestations, the age peculiarities in the course of infections, etc. The therapy of acute infections was also insufficiently effective.

The many years of experience in treating infections with serums have shown that, however valuable, this method failed to solve the problem of treating infections.

All the aforesaid emphasised the urgent necessity of new approaches to the study of acute infections.

A new and broader trend in studying infectious diseases came into existence in the 1930's and has since gained wide currency mainly owing to the works of Soviet scientists—clinicians and theoreticians. This trend may be named the pathogenetic trend. Soviet scientists have stressed the very important role of the macroorganism, its nervous system and reactivity in the emergence and course of the infectious process; at the same time they have not denied the significance of the causative agent of the disease, but considered it in its interrelations with the reactions of the organism and the conditions of the external and internal environment.

This has ensured a deeper understanding of infectious disease as a dynamic process operating cyclically with a successive alternation of periods (phases), each phase characterised by a definite functional state of the nervous system, the vegetative nervous system in particular.

Clinical and experimental research has established that a so-called allergic state (i.e., altered reactivity of the organism) develops in the course of an infectious disease owing to increased sensitivity of the organism to the products of splitting of the microbial proteins. The allergic reactions which take place at this time and are in many respects similar to the serum sickness (which is a reaction of the organism to the administered protein of a foreign serum) play an important part in the pathogenesis and clinical aspects of infectious disease; they condition the fluctuating course of the process and reduce the patient's resistance and immunity, thus paving the way to emergence of complications, relapses of the disease, intercurrent secondary infection, etc.

These allergic reactions most frequently occur in the second, third and fourth weeks of the disease (as they say, "during its second half", during the allergic period), although under certain conditions they manifest themselves earlier, during the incipient stage of the disease. This applies particularly to the gravest—hypertoxic—forms of acute infections in whose pathogenesis the significance of the increased sensitivity of the organism at the time these forms of disease develop has been demonstrated (clinically, anatomically and experimentally).

These facts clearly show the role of the macroorganism in the formation of infectious disease.

The deepened knowledge of the pathogenesis of infections and of the laws of its operation has served as the basis for the successful elaboration of the therapy of acute infections. The studies have revealed a necessity for a pathogenetic therapy of infections; the treatment must be administered strictly individually, according to definite clinical indications and with due regard for the phase of the process, the age of the patient and the state of his reactivity.

Pathogenetic therapy includes the specific method directed against the causative agent and its toxins, as well as the microbes of the secondary complications (treatment with serums, sulfa drugs, antibiotics—penicillin, synthomycin, streptomycin, etc.), and the methods which favourably influence the reactivity of the organism and the patient's emotional tone (blood transfusion, administration of blood plasma and serum, gamma globulin, physiotherapy, etc.).

In addition, the complex of pathogenetic therapy includes, as one of the most essential factors of favourable influence on the organism in its struggle against infection, setting up a hygienic atmosphere for the patient, good care and a rational diet. The necessity of maximal attention to this aspect of treatment must be emphasised in every possible manner.

The principles of pathogenetic therapy, which have become deeply rooted in the work of Soviet pediatric institutions, have greatly contributed to the reduction of mortality from infections.

The development of medicine and its reorganisation on the basis of Pavlov's physiological teaching offer extraordinary prospects for further fruitful study of acute infectious diseases and for decisive achievements in the control of these diseases.

The studies of acute infections conducted in this direction have only just begun. They are based on the fundamental propositions of Pavlov's teaching on the integrity of the organism, the unity and constant interaction of the organism with its environment, and the leading role of the nervous system in the activity of all organs regulated by the cerebral cortex and its subordinate centres, which effect this regulation by means of reflexes through the nervous apparatus (receptors) found in all organs.

These studies show that disturbances in the activity of the nervous system play a determining role in the origin and course of infectious processes and that the nervous system plays an organising role in the process of recovery.

Microbes and toxins are regarded as specific stimuli which by active contact with the nervous apparatus (receptors) evoke a reflex reaction; the stimulation affects the central nervous system

and through it the organism as a whole, producing a definite picture of the disease that corresponds to the specific nature of the given causative agent.

The state of the central nervous system and the degree of its excitability are in large measure responsible for the emergence of infectious disease, its gravity and course.

The first to be affected by the action of toxins through the receptors is the cerebral cortex. The earliest signs of intoxication are a result of its inhibition in response to the infectious influence: the cells of the cortex are a safeguard against activity under harmful conditions (protective inhibition). The underlying subcortical vegetative centres are freed against the background of cortical inhibition and constitute the main force in fighting the infectious and toxic principle. During the different periods of the subsequent operation of the process changes take place in cortical and its subordinate vegetative reactivity.

In the struggle against infection the defensive reactions in the integral organism manifest themselves not only in the formation of various specific antibodies and mobilisation of the means of natural (physiological) immunity, but also in a number of other functions; the central nervous system participates in all of them through complex reflex reactions.

The changes in reactivity in the course of acute infections and the onset of an allergic state, i.e., hypersensitiveness, are also largely connected with the changes in the nervous reflex regulation of the processes in the organism.

Pavlov's teaching on nervism warrants a new interpretation of a number of clinical facts.

The age peculiarities in infections, particularly in early childhood, find their explanation in the peculiarity of development and functioning of the children's central nervous system; it is precisely because of the incomplete development of the higher parts of the nervous system, especially the cerebral cortex, and the incomplete reflex activity connected with it that young children are inclined to respond with general (diffuse) reactions to the most diverse harmful influences.

Cardiovascular disorders which so often occur in acute infections ("infectious heart") are for the most part conditioned by disturbances in the nervous regulation of the circulatory system and only in some infections (especially diphtheria) is the heart itself (the heart muscle) affected.

And, lastly, the relapses of infectious disease, the prolonged course of some infections and the protracted functional disorders

which sometimes persist after these infections are largely due to disturbances in the trophic influence of the central nervous system and to the action of the foci of excitation retained in it, these foci of excitation conditioning the so-called trace reactions.

The aforementioned principles of pathogenetic therapy remain valid, but they must be employed primarily to exert an effective influence on the patient's central nervous system. It is necessary carefully to consider the patient's neuropsychic state and behaviour, to safeguard his peace of mind and sleep, to perform the fewest required traumatising (painful) manipulations, and to help in enhancing his emotional tone by kind and considerate treatment.

The patient must be kept as much as possible outdoors because of the salutary effect of fresh air on the central nervous system; the sick child must be ensured a hygienic atmosphere and good care and must be given a variety of nourishing foods; no efforts must be spared to improve the child's appetite.

The aggregate of all these measures, in addition to specific therapy and special methods of treatment, can best ensure complete recovery of children from infectious diseases.

SCARLET FEVER

Etiology. The causative agent of scarlet fever is unknown. The various views regarding its etiology can generally be reduced to two principal theories, namely, the streptococcus theory and the filtrable virus theory.

According to the streptococcus theory, the causative agent is a hemolytic streptococcus which produces a specific toxin.

This theory was advanced as early as the 1890's and Russian scientists greatly contributed to its development. Thus G. Gabriчевsky was the first to propose and carry into effect antiscarlatinal inoculations with a vaccine prepared from streptococcus cultures taken from scarlet fever patients. I. Savchenko was the first to isolate the toxin of the scarlet fever streptococcus and to study it experimentally.

The subsequent works of George and Gladys Dick (American physicians, 1920's) supplemented the theory of the streptococcus etiology of scarlet fever. They proposed: a) an intracutaneous test with the toxin of a hemolytic streptococcus to determine susceptibility to scarlet fever, b) a method of prophylactic

inoculations using this toxin, and c) a curative antitoxic serum.

All the above has been widely used in the control of scarlet fever. The question of the etiology of scarlet fever has been very intensely studied in the Soviet Union during the last 15-20 years. Some scientists (M. Danilevich, V. Ioffe et al) are inclined to consider the streptococcus etiology of scarlet fever adequately established. While recognising the *important role of the streptococcus in the pathogenesis and course of the disease*, many scientists hold, however, that its true causative agent belongs to the group of so-called filtrable viruses. This view, also long since advanced (40 years ago), still has many advocates, although it has not been definitely confirmed.

It is quite probable that scarlet fever is really caused by a filtrable virus which, however, gains entrance into the organism together with the streptococcus and that there is thus an intimate connection between them. This latter theory (virus+streptococcus) explains, better than the other theories, many clinical aspects of the disease.

Epidemiology. Ways of transmission of the infection. The disease may be transmitted from a person affected with the disease to a healthy individual by *direct contact or by means of droplets of moisture containing the causative agent* (during coughing, sneezing or talking), through various *things*—clothing, toys, books and foodstuffs (for example, milk)—infected by the patient, and through *carriers of bacilli*, i.e., healthy persons who having been in contact with patients harbour the causative agent and thus spread the disease (this must be constantly remembered by the medical personnel of scarlet fever wards).

Unidentified mild atypical cases in which the patients have not been isolated, constitute a particular epidemiological danger.

This applies not only to children, but also to adults in whom the course of the disease may simulate that of sore throat without the eruption characteristic of scarlet fever. This explains why in some cases of scarlet fever it is impossible to establish the source of infection.

Scarlet fever patients are contagious as long as they are sick and possibly also during the last days of the incubation period. All of the patient's excretions (sputum, saliva, urine, desquamating epithelial scales, and mucus from the nose and fauces) are contagious.

The period during which a scarlet fever convalescent continues to be contagious cannot be considered definitely established.

Despite the definite parallelism between the period during which the hemolytic streptococcus is retained in the convalescent's fauces and his epidemiological danger as a source of infection, examinations of the fauces and nasopharynx for the presence of hemolytic streptococci cannot serve as a very reliable criterion for the solution of this problem (the assertions of some authors to the contrary notwithstanding). The pathological state of the fauces and nasopharynx, and the presence of complications (especially purulent) apparently play the most important part in spreading the infection.

Until very recently isolation of scarlet fever patients for 30-40 days from the onset of the disease was current in medical practice. This period was set up empirically, without sufficiently valid reasons, and did not always exclude the danger of transmission of the infection by convalescents discharged from the hospital. Communication of the disease by these convalescents to those in their immediate surroundings (so-called late contacts) was observed in 3-5 per cent of the cases even when the patients had been confined to hospital for more than 40 days.

It was thus found that in controlling the contagiousness of scarlet fever convalescents it was not increasing the period of hospitalisation, but rather improving the quality of hospitalisation that produced better results.

In accordance with this, considerable changes have been introduced in recent years into the methods of hospitalisation of scarlet fever patients and their discharge upon recovery.

Man is less susceptible to scarlet fever than to measles so that even during a scarlet fever epidemic only a moderate percentage of the formerly unaffected population contracts the disease.

Children particularly frequently contract scarlet fever between 18 months and 10 years of age. Infants between three and six months of age are affected by this disease much less frequently because they are isolated from the environment and have acquired immunity through the blood (placenta) of their mothers during intrauterine life and through their mothers' milk.

Scarlet fever is observed in large cities in the form of separate foci; within certain intervals of time (5-10 years) it produces epidemics of varying intensity and gravity.

The disease occurs most frequently in autumn and winter because during these seasons the child spends less time outdoors and more often comes in contact with patients.

An attack of the disease confers lifelong immunity, although in rare cases scarlet fever nevertheless recurs.

Pathogenesis. Many aspects of the pathogenesis of scarlet fever are still unclear. It is questionable whether scarlet fever is a disease in which the microbe is in the blood or the causative agent remains, as in diphtheria, only in the pharynx, while the organism is affected by the toxin which is transported by the blood stream. The latter view is advocated by the supporters of the streptococcus theory of scarlet fever who believe the hemolytic toxin-producing streptococcus to be this local (living in the fauces) causative agent. They explain the fundamental picture of the disease—pyrexia, eruption and neurovascular symptoms—by the action of the toxin, and the complications, especially the purulent complications—by penetration of the streptococcus into the tissues. George and Gladys Dick proposed a skin test, analogous to the Schick test for diphtheria, consisting of an intracutaneous injection of 0.2 ml of streptococcus toxin (Dick test). People who have no antibodies (antitoxins) in the blood *respond with a positive reaction* in the form of an inflammation—red papule—at the point of injection. If the people injected the above toxin have antitoxin in their blood, the antitoxin neutralises the toxin, the inflammation does not develop and a *negative* reaction is observed. This test reveals the children-susceptible to the infection.

In persons who have recovered from scarlet fever the Dick test changes from positive to negative, which, however, is not always the case.

Other authors hold that the streptococcus is the microbe which causes only the secondary complications, whereas the picture of the disease is produced by the action of a specific *causative agent* which circulates in the blood; this agent is classified with the group of so-called *filtrable viruses* which are in some manner possibly related to the streptococcus. According to this opinion, scarlet fever is a *general blood infection*.

Two aspects—toxic and *septic*—strike the eye in the course of the disease.

The toxin produced by the causative agent of scarlet fever acts very selectively on the neurovascular apparatus, frequently provoking severe nervous disorders during the acute stage of the disease, and during the second week—a very peculiar picture of cardiac disorders (scarlet fever heart). This is the toxic aspect of the process; the disease is additionally attended with an easily and rapidly developing local process in the form of local necrotic angina, affection of the cervical glands and emergence of pyoseptic complications.

Lastly, some authors hold that an important part in scarlet fever is played by preliminary microbial sensibilisation of the organism, which renders the latter *extraordinarily sensitive to the streptococcus*; the picture of the initial stage of scarlet fever, which somewhat resembles the serum sickness, develops in response to the penetration of the streptococcus (allergic and anaphylactic theory of scarlet fever).

This theory fails to explain the principal questions of epidemiology and immunity in scarlet fever (namely, its contagiousness and the permanence of immunity developing after recovery from scarlet fever).

The latter view is of some interest in that it helps to account for the pathogenesis of scarlet fever, especially its second stage.

Today most researchers believe that this modified sensitivity accounts only for the extraordinary regularity of the complications (nephritis, lymphadenitis), which appear during the “second stage of scarlet fever, and sometimes for the “recurrence” of the main symptoms of the disease (eruption, sore throat). Thus a *third aspect* connected with the influence of allergic mechanisms* must also be distinguished in the pathogenesis of scarlet fever.

* Allergy is an altered reaction capacity.

It would be more correct, however, to think that the allergic state of the organism is conditioned by the virus itself from the moment it has gained entrance into the organism, rather than by a preliminary sensibilisation by the streptococcus. The viruses (smallpox, influenza, etc.), as we know today, are themselves potent allergens.

This does not, of course, exclude the fact that streptococci, which play so important a part in the pathogenesis and course of scarlet fever, also have something to do with the emergence of allergic reactions.

Pathological anatomy. The primary localisation is characterised by inflammation in the fauces, pharynx and nasopharynx—catarrhal in mild cases and necrotic in severe cases. Such necrosis not infrequently gives rise to disintegration and ulcers which heal slowly with formation of cicatricial tissue. The respiratory tract—nose, larynx and trachea—are affected much more seldom, but the character of the changes is the same.

The adjacent cervical glands also exhibit symptoms of inflammation and often of suppuration. In the gravest cases mortification (necrosis) involves the surrounding cervical glands and cellular tissue, producing a picture of a so-called *dry cervical phlegmon*.

In the heart—symptoms of albuminous and fatty degeneration of the muscle and myocarditis (in severe septic forms).

Acute endocarditis is a very rare occurrence in scarlet fever. It is usually of septic origin. It is observed together with other septic manifestations, particularly purulent pericarditis, and quickly leads to death.

The endocarditis which sometimes develops during and after scarlet fever and leads to *cardiac failure* is, as present-day research has shown, rheumatic. Scarlet fever can aggravate and activate latent rheumatic infection. A considerable part in the genesis of such aggravations is played by allergic mechanisms.

Nephritis is predominantly an affection of the vascular apparatus of the kidney (acute glomerulitis, septic and interstitial nephritis); it occurs alone or more frequently is attended with nephrosis, a degenerative change in the convoluted tubules. Such diffuse affection of the kidney is usual and most typical of scarlet fever.

In severe cases of scarlet fever sections always show destructive changes in the ganglia of the vegetative nervous system; degenerative changes in the cells of the cerebral cortex and subcortical ganglia have also been described.

Otitis, mastoiditis, purulent lymphadenitis, pneumonia and purulent pleurisy are the *other complications* observed in scarlet fever.

The gravest picture is that of general streptococcal sepsis with purulent foci in the internal organs, purulent affection of the joints and sometimes purulent meningitis. In recent years grave complications of scarlet fever have been very rarely observed.

Clinical findings. The incubation (latent) period of the disease lasts an average of 4-6 days; sometimes it takes only a few hours, but in some cases it is prolonged to 11 days. During this period there are no clinical manifestations.

Onset and course. The disease most frequently begins with a sharp rise in temperature to 39-40°, nausea, vomiting, headache and, often, chills and pain during swallowing (in older children). Infants, and in severe cases also older children, may exhibit cere-



Fig. 45. Eruption in Scarlet Fever

bral symptoms—convulsions, delirium, sleepiness and clouded consciousness. In most cases an examination of the patient at that time still shows the skin to be clear, while a bright, often *punctate redness* is already discernible in the *fauces* (soft palate and uvula) and is demarcated along the border of the hard palate.

Within a few hours, but more often at the end of the first or in the beginning of the second day, an eruption appears; in typical cases of scarlet fever its main signs are usually clearly defined on the second or third day; the most characteristic signs are *eruption*, *affection of the fauces* and *intoxication* (general poisoning) (Fig. 45).

The *eruption* starts on the neck and upper part of the chest and back, then quickly spreads over the entire body and attains its full development for the most part on the second or third day. It is most clearly defined on the medial and flexor surfaces of the limbs (in the elbow, inguinal and popliteal regions), the anterior and lateral parts of the chest and the lower half of the abdomen (in these places it is usually most clearly defined even when it is barely discernible in other places). The character of the eruption varies; sometimes it is a mere redness of the skin—*erythema*, but more often it is from the very outset a *punctate eruption against an erythematous background*. In grave cases the eruption may assume the character of *large dots*, even spots, and sometimes may be slightly exudative.

The eruption may be of different shades, depending on the gravity of the case—from pink to saturated red, and in severe cases cyanotic. Sometimes it is attended with profuse hemorrhages into the skin. Separate hemorrhages, especially in places of flexure, are often observed in moderate cases.

The eruption is not infrequently attended with pruritus.

If you draw a finger along the skin of a scarlet fever patient without exerting particular pressure, you will observe within a few (7-8) seconds a clear white line (white dermatographism) owing to which the eruption can be very well seen. In grave cases this "white line" is either very *feebly* defined or it *does not appear* at all, which is of great prognostic value since it attests considerable intoxication.

The *face* of the scarlet fever patient is quite characteristic: the middle of the face—the lips, nose and chin—remains white, sharply differing from the red cheeks (*scarlet fever triangle*).

Examination of the pharynx at once reveals a bright colouring of its mucosa—succulent, bright *angina* ("blazing fauces"). In mild cases it is catarrhal, in moderate cases it is follicular-lacunar or—

usually not until the second or third day of the disease—necrotic (moderately). Severe cases are characterised by vast, foul, deep necrosis which involves the fauces, tonsils, lateral and posterior walls of the pharynx and the nasopharynx. Corrosive discharges often appear from the nose in connection with the severe necrotic process in it.

The *tongue* is very typical; during the first two or three days it is dry and coated with a white film; from the third day on the film disappears from the tip and edges and the tongue becomes hyperemic with very prominent papillae (strawberry tongue).

The *cervical glands* which collect the lymph from the fauces and pharynx (tonsillar) enlarge (moderately in catarrhal and considerably in necrotic angina); in more severe cases the tissues surrounding them become inflamed (periadenitis) and in the gravest cases necrotise (adenophlegmon); in these cases a tumour—very painful, red and hard as a board—develops on the neck. Subsequently the skin under the tumour liquefies, and a vast section of the tissues on the neck disintegrates and exposes the muscles and vessels, thus threatening hemorrhages.

Of late there have been very few such cases, but before the Second World War they occurred more frequently and often ended lethally.

At the height of the eruption all peripheral lymph nodes become somewhat enlarged—sometimes as large as peas and succulent.

Intoxication (general poisoning) manifests itself in pyrexia (39-40°), headache and jadedness. Weakness, delirium, unconsciousness, sometimes convulsions and meningeal symptoms (occipital rigidity, red dermographism and Kernig's sign) are observed in severe cases. The pulse is quick and does not correspond to the temperature (140-180 beats per minute). The blood pressure is elevated, the skin is dry, and perspiration is absent. No particular changes in the organs are observed during this period.

Only slight pulmonary emphysema is noted (the relative border of the heart is not clearly defined and there is hepatic dullness at the 7th or 8th rib).

Less urine is excreted but the urine is saturated. Sometimes it contains albumin, casts and an increased amount of urobilin.

The blood shows a high leukocytosis (up to 20,000-30,000 and more leukocytes) with neutrophilia and a change in the band cells, as well as an absence of or decrease in eosinophils which on the third or fourth day begin to increase. The erythrocyte sedimentation rate is high (30-60 mm per hour).

The intestines usually function normally; sometimes the patients are constipated; only nurslings, and in very severe cases older children, develop diarrhea.

In nurslings the process not infrequently operates atypically; the eruption is not clearly defined, and the intoxication manifests itself in that the child is very restless and refuses the breast.

Such are the symptoms of scarlet fever at the height of the process (on the second or third day) in typical cases of moderate severity.

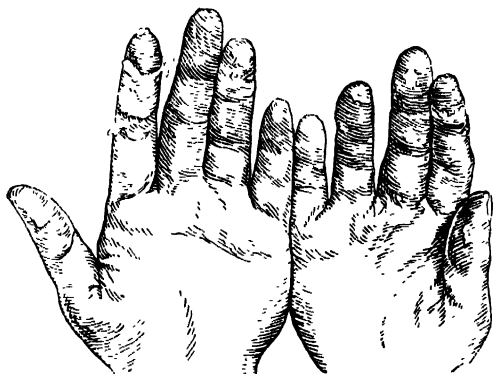


Fig. 46. Scaly desquamation on fingers in scarlet fever

On the fifth or sixth day of the disease the symptoms start diminishing.

The *eruption* begins to fade and disappear, leaving a negligible pigmentation only in the cases in which it was *strongly pronounced and saturated*.

Extraordinarily typical of scarlet fever is desquamation which usually begins in the second week of the disease. The desquamation is *scaly*. Usually it starts at the tips of the fingers (Fig. 46) and toes, and sometimes involves the entire palm of the hand and sole of the foot. It ends in the sixth or seventh week of the disease. In cases of an abundant and, especially, miliary eruption (with elements in the form of little whitish vesicles with a serous content) the desquamation may begin earlier, sometimes even before the disappearance of the eruption, and may involve the trunk.

The changes in the fauces disappear at different periods of time, depending on the form of the disease: catarrhal and follicular anginas disappear on the fourth, fifth or sixth day, while necrotic

anginas may last very long, simultaneously with irregular high temperature and lymphadenitis, giving complications in the form of ulcers, lateral pharyngeal and posterior pharyngeal abscesses which heal slowly and sometimes serve as a source of sepsis.

In cases of moderate severity the *intoxication* disappears on the fourth or fifth day; the temperature drops sometimes rapidly and sometimes more slowly, the headache passes, the patient regains his appetite, the leukocytosis and neutrophilia decrease, and eosinophils appear. Sometimes these changes occur even earlier—on the third day—and quite rapidly, so that by the beginning of the second week, in moderately severe cases, the child has a normal temperature, his general condition considerably improves and he looks well. During this period, however, very characteristic cardiac symptoms, referred to as a “scarlet fever heart” develop. The pulse becomes *slow* (bradycardia) and *irregular* (arrhythmia), the heart somewhat dilates, the first heart sound ceases to be clear, sometimes there is a systolic murmur, the blood pressure drops and the liver becomes enlarged, protruding 2-4 cm from under the hypochondrium. This state continues for up to three weeks and then gradually passes without imperilling the patient's life or requiring any special treatment, except rest.

As regards the essence of the “scarlet fever heart”, thorough studies (clinical, electrocardiographic and radiologic) do not warrant the assumption of myocarditis as the basis of its manifestations.

The cause of these disorders mainly consists in a disturbance in the nervous regulation of the cardiovascular function under the influence of the toxin, which also affects the functions of the heart muscle (vegetative-metabolic disorders, “infectious heart”).

Myocarditis is observed only in severe septic cases of scarlet fever. Cardiovascular disorders arising during the allergic stage of scarlet fever and corresponding in their manifestations to the picture of mild myocarditis have been described in recent years under the designation of “allergic myocarditis”; these disorders run a favourable course.

Very seldom septic pericarditis and endocarditis may arise during scarlet fever as manifestations of septicopyemia.

Endocarditis, which sometimes develops in the course of scarlet fever, during its second, allergic stage or soon after recovery from scarlet fever and ends in heart disease, owes its origin not to scarlet fever but to an aggravation of a rheumatic infection which until then was latent.

If no complications develop, the patient begins to feel good in the third week and may be allowed out of bed. However, this is not always the case, and scarlet fever often gives rise to a number of complications.

Early complications. These complications sometimes appear during the very first days of the disease.

Lymphadenitis. The glands enlarged during the first days usually begin to contract to normal size within 2-4-5 days and then suddenly start greatly enlarging again, become painful and cause a considerable rise in temperature.

This may continue for a number of days, but the whole thing ends either in resorption (*simple lymphadenitis*) or in purulent liquefaction (*purulent lymphadenitis*) which requires an incision. Such lymphadenitis is caused by penetration of the *streptococcus* which is usually found in the pus.

Simple or suppurative otitis (inflammation of the middle ear) may also develop during the first days of the disease. The signs are pain and pyrexia, and in suppurative otitis—*otorrhea* (discharge from the ears).

Suppurative otitis may last very long; it often impairs hearing and in cases of severe necrotic injuries to the middle ear with destruction of the auditory ossicles the process extends to the internal ear, causing grave impairment of hearing and sometimes total deafness.

In other cases the process extends from the cavity of the middle ear to the accessory cavities, involves the cells of the tympanic antrum and the mastoid cells, which gives rise to *mastoiditis*, a complication that may require surgical intervention (trephining of the mastoid process). In virtue of all this, inflammation of the ear is a very dangerous complication of diplostreptococcal etiology and may serve as a source of septicopyemia. It is therefore necessary carefully to watch its course and administer vigorous treatment (see below).

Sinusitis—ethmoditis, frontitis and highmoritis—(inflammations of the accessory cavities of the nose) are some of the early complications which may be observed in scarlet fever.

Ethmoiditis is characterised by intense discharge from the nose, edema and pain in the region of the eye, and protrusion of the eyeball; *frontitis*—by edema of the bridge of the nose, forehead and the supraorbital ridges; *highmoritis*—by swelling and pain in the region of the cheek and lower eyelid.

Retropharyngeal and lateropharyngeal abscesses, as well as sto-

matitis—aphthous, ulcerative and necrotic—is often observed in very young children.

Pneumonias, usually microfocal, are observed during the first weeks of scarlet fever relatively rarely mainly in very young children and most frequently in combination with influenza or in severe septic forms of scarlet fever. Pneumonias are often attended with purulent *pleurisies* which may also be primary. The pneumonias and pleurisies are usually of diplostreptococcal etiology.

Synovitis (serous inflammation of synovial membranes of joints) is a benign complication which develops for the most part at the end of the first and in the beginning of the second week of scarlet fever; it is characterised by a painful swelling mainly of the small joints (of the hands, feet, fingers and toes, and more rarely of other parts) and pyrexia. These symptoms are unstable and retrogress spontaneously; toxic and allergic factors play some role in their genesis.

Scarlet fever runs multifarious courses and is a very dangerous disease; sometimes, even in relatively mild cases, the disease may take a graver course on the fourth or fifth day; numerous purulent complications may develop early and in the end lead to septicopyemia, i.e., the appearance of the streptococcus in the blood and its localisation in the organs. Clinically septicopyemia most frequently manifests itself in a purulent affection of the joints and in septic interstitial nephritis.

Sluggish necrotic processes in the fauces, pharynx and nasopharynx, suppurative otitis and latent mastoiditis are the causes of early sepsis.

Affections of the *larynx* and *trachea*, which produce symptoms of stenosis (croup) are rare in scarlet fever. They occur more frequently in severe cases and in very young children, particularly in combination with influenza

Late complications. In the second week the patient affected with a moderately severe form of scarlet fever not infrequently appears perfectly well, i.e., the temperature is normal, the patient has regained his sleep and appetite, and there is only desquamation of the skin on the arms and legs and insignificant remains of cardiac symptoms. However, this condition must not as yet serve to reassure the physician. In quite a few cases so-called *late complications* suddenly develop in the third week (between the 15th and 20th days) and sometimes later; these complications are characterised by a new rise in temperature, a change in the patient's general condition for the worse and sometimes even reappearance

of the symptoms of the incipient stage, i.e., *angina* and *eruption* (so-called relapse of scarlet fever).

A peculiar syndrome referred to as an "allergic wave" not infrequently emerges in the third or fourth week of scarlet fever. The syndrome manifests itself in pyrexia, quick pulse, low blood pressure, enlargement and succulence of the peripheral lymph nodes, and appearance of unstable eruptions of a macular and urticarial character. In some cases these symptoms disappear rather rapidly; sometimes the allergic wave is followed by complications.

The most typical of these complications are *nephritis* and *lymphadenitis* which often take place simultaneously. In addition to the appearance of pyrexia, the beginning of the development of nephritis is often characterised by nausea or vomiting and diminished excretion of urine which grows dark, *saturated* and not infrequently soon becomes *sanguineous*. At the same time (sometimes even sooner, in other cases somewhat later) *general* edema develops. The face grows pale and puffy, edema of the limbs and trunk appears and the severest cases are attended with cavitory dropsy (fluid is found in the abdominal, pleural and pericardial cavities).

Systematic *weighing* of the patient very soon and precisely reveals retention of water and an increase in weight.

Analysis of the urine shows the latter to contain *albumin*, sometimes a very large number of different forms of *casts*, leukocytes and erythrocytes; the specific gravity of the urine increases.

Nephritis is not only a renal affection. It is a disease of the entire organism, greatly affecting the vessels, especially the capillaries, disturbing the metabolism (in the first place the water-salt and nitrogen) and changing the composition of the blood, i.e., its content of proteins and their fractions, as well as other constituents.

The most pronounced in the clinical picture of acute scarlatinal nephritis is the cardiovascular syndrome; elevation of the blood pressure is one of the very early and constant symptoms connected with the general spasm of the vessels; the heart sounds are intensified, especially the second sound on the aorta; sometimes there is a murmur and dilatation of the heart. The pulse becomes tense and slow. In severe cases, in connection with the development of considerable edema and cavitory dropsy, the work of the heart is greatly hampered and the patient may die of cardiac weakness, mainly myocardial insufficiency brought about by degenerative, inflammatory and ischemic processes in the heart muscle.

The most important symptoms attesting the developing myocardial insufficiency are tachycardia, gallop rhythm, dyspnea, cyanosis and enlargement of the liver.

In grave cases the urine may cease to be secreted altogether (anuria) and the organism is poisoned with the unexcreted metabolites, which leads to uremia.

In cases of retention of sodium chloride in the tissues and blood, and in cerebral edema, attacks of *clonic* spasms follow each other and the patient loses consciousness—*uremic eclampsia*, which not infrequently, however, may pass within a few minutes or hours. Sometimes it may lead to temporary *blindness* (amaurosis).

More dangerous is the so-called true—azotemic—uremia associated with insufficient excretion of *protein metabolites* through the kidneys and characterised by headache, coma, odour of urine from the mouth, extraordinarily high blood pressure and low specific gravity of the urine. Examination of the blood shows increased residual nitrogen (the norm for children is 30-40 mg%). Azotemic uremia usually ends lethally. It very rarely occurs in acute nephritis, but takes place mainly in *chronic* renal disease.

Nephritis lasts 2-4 weeks and may end in complete recovery; it seldom persists for about three months and uncommonly rarely develops into a chronic form. Death in cases of nephritis may be due to *uremia*, increasing *dropsy* of the abdominal and other cavities, cardiac insufficiency, *pneumonia* and purulent complications.

Nephritis is not infrequently attended with simple lymphadenitis, when parallel with nephritis the lymph nodes greatly enlarge in the third week. Suppuration is rare. Sometimes such lymphadenitis develops alone, without nephritis, in the third week. Other complications, such as otitis and affections of the joints are also observed during the late stage of the disease.

The altered reaction capacity of the organism, the state of increased sensitivity and allergy are considered to be the causes of such late complications following a period of temporary well-being. These complications and the attending eruption and angina are ascribed to the increased sensitivity of the organism, acquired during the first stage of scarlet fever.

Forms of scarlet fever. Scarlet fever yields a great variety of forms differing in the severity of their manifestations and course; in addition to the mildest forms with an abortive course and the so-called effaced forms which are difficult to diagnose, there are extraordinarily grave hypertoxic forms which end lethally within

one or two days (so-called fulminant scarlet fever). In a clinical classification of all these forms of scarlet fever it is necessary to consider their type, severity and course (see classification scheme on p. 390).

In *typical* forms all the basic signs of scarlet fever are clearly defined; in *atypical* forms one of the cardinal symptoms of the disease—eruption or sore throat—is either not clearly defined or is entirely lacking. These are the so-called effaced forms of scarlet fever and extrabuccal scarlet fever (traumatic, burn). Atypical scarlet fever also includes the hypertoxic cases in which general intoxication is very strongly pronounced, while sore throat and eruption are barely discernible.

In determining the *severity* of scarlet fever the degree of general intoxication and the intensity of the local process (in the fauces, nasopharynx and glands) are mainly taken into consideration. In determining the *course* of the disease the presence or absence of complications or fluctuating flare-ups of the process and their character are taken into account. The forms characterised in the main by a picture of severe intoxication of the organism are considered toxic, while those in which a severe local process with numerous early complications and development of sepsis comes to the fore are regarded as septic.

In *mild forms of scarlet fever* intoxication is insignificant, the temperature is not high, angina is catarrhal, the glands are but slightly enlarged and the eruption is benign. All the symptoms regress rapidly. Purulent complications are rare. Late complications, particularly nephritis, are possible.

In *moderately severe forms of scarlet fever* intoxication and the local process in the fauces are well pronounced but are of moderate intensity, the temperature is high, the eruption is uniform with good vascular reaction and distinct white dermographism, angina is lacunar or necrotic, but the necrosis is not widespread, and not deep; there is also a moderate regional lymphadenitis.

Danger may arise only at a later stage of the disease with the development of complications which are more frequent than in mild forms (nephritis, lymphadenitis, purulent complications and sometimes general septic complications).

Severe forms. The most prominent feature in the clinical picture of severe toxic forms of scarlet fever is the rapid (sometimes within several hours) development of general intoxication with acute affection of the nervous system and the cardiovascular apparatus, seriously imperilling the patient's life. In cases in which

Classification of Scarlet Fever

Type	Severity	Course
I. Typical forms	I. Mild forms a) transitional (A, B, C) to moderate severity	I. Without allergic waves or complications
A. With predominance of toxic symptoms (toxic forms)	II. Forms of moderate severity a) transitional (A, B, C) to severe	II. With allergic waves and complications: a) allergic type (nephritis, synovitis, reactive lymphadenitis);
B. With predominance of the local process	III. Severe forms Indices of severity: General: a) meningoencephalitic syndrome; b) early metabolic-vegetative syndrome (early sympathicoparesis); c) hemorrhagic syndrome; d) early manifestations of septicopyemia	b) purulent; c) septicopyemia
C. Mixed forms		III. Abortive course
II. Atypical forms		
D. Forms with aggravated symptoms:		
a) hypertoxic forms;		
b) hemorrhagic forms		
E. Effaced forms (scarlet fever without eruption)		
F. Extrabuccal forms (traumatic, burn)		

these manifestations are very strongly pronounced the toxic forms lead to collapse and death (hypertoxic, fulminant scarlet fever) within the very first day or two. The symptomatology of toxic scarlet fever is essentially characterised by a violent onset, very high temperature, repeated pernicious vomiting, restlessness, delirium, impaired consciousness or coma, meningeal symptoms, adynamia, narrow pupils, absence of abdominal reflexes, injection of the scleras, scanty and uneven cyanotic eruption with hemorrhages, absent or barely discernible white dermographism, very sluggish vascular reaction of the skin, very rapid and weak pulse, embryocardia, weakened heart sounds, and low blood pressure. The changes in the fauces and glands may be slight. Frequent, liquid, green stool is sometimes observed.

Thus, the afore-described picture shows stimulation or inhibition of the central nervous system and affection of the vegetative-endocrine apparatus, which accounts for the development of the severe cardiovascular insufficiency.

The *hemorrhagic form* of scarlet fever which, in addition to the severe general symptoms, is characterised by numerous hemorrhages into the skin is the gravest and most dangerous. The outcome is usually lethal.

Severe toxic forms of scarlet fever are observed mainly in older children and only as an exception in very young ones.

Septic forms occur more frequently in young children and are characterised by rapidly developing vast and deep necrosis in the fauces, extensive lymphadenitis and *adenophlegmon*. There are numerous and mainly purulent complications (otitis, lymphadenitis, mastoiditis, purulent arthritis, pneumonia, purulent pleuritis, septic nephritis, and general septicemia and septicopyemia). *Scarlet fever antitoxic serum* has but little effect in septic scarlet fever.

Vigorous penicillin therapy, blood transfusions and various forms of hemotherapy are indicated.

The *effaced forms* are distinguished by the fact that one of the main symptoms—eruption or angina—at times, both) is either absent or insufficiently clearly defined. Of late these forms have occurred rather frequently and are very difficult to diagnose. However, by careful examination it is possible to detect barely discernible traces of eruption on the skin and changes in the mucosa.

Anamnestic data indicating contact or a focus in a children's institution, and the mother's statement concerning an appearance and disappearance of a rash are very important.

Extrabuccal scarlet fever (traumatic, burn) is characterised by a short incubation period (1-2 days), special development of the eruption which usually begins at the site of injury or burn, absent or feebly pronounced angina (necrotic angina suggests that the scarlet fever case is not of extrabuccal origin). This form of the disease for the most part runs a mild course and, if complications do occur and cases with a severe course and lethal outcome are observed, we have to take into consideration the part played by the primary affection (vast burns are not infrequently themselves responsible for considerable intoxication and are attended with various septic complications).

Scarlet fever peculiarities in recent years. In recent years the incidence of scarlet fever has been characterised by noticeably diminished severity of the initial manifestations of the disease, its mild course, and considerable decrease in the number of complications. The severity of scarlet fever, which began to decrease in the 1920's, diminished particularly during the 1940's-1950's. During the 1920's-1930's mild forms constituted 20-30 per cent of the disease incidence, whereas in 1950 they amounted to 80-82

per cent (as evidenced by the data of Moscow and Leningrad hospitals). Severe toxic and septic forms now occur as a rare exception. Many cases of effaced scarlet fever and scarlet fever with an abortive course are observed. In recent years the total number of complications has not exceeded 10-15 per cent, while that of purulent complications has been reduced to a fraction of one per cent. The complications observed today are for the most part simple lymphadenitis and catarrhal otitis; scarlatinal nephritis is rare and, as a rule, it runs a mild course. Since 1949 scarlet fever mortality has been reduced to zero. The mild course of scarlet fever may be due to the improvement in the health of the child population occasioned by the steady rise in the standards of living of the working people and the achievements of Soviet medicine, particularly the quality of hospitalisation and regimen, as well as the elaboration and employment of new effective methods of treatment (penicillin therapy), prevention of disease, etc.

Prognosis. The probable course and termination of scarlet fever must be forecast with great caution.

The severity of the disease is not always determined during its first days; sometimes scarlet fever is of moderate severity at the outset, but may take a severe course on the fourth or fifth day.

It is very difficult to tell *whether or not complications will develop* and what their outcome will be. At any rate the disease does not warrant any self-assurance until the end of its third week (even mild cases of scarlet fever are not guaranteed against late complications).

Cases in which violent symptoms on the part of the nervous system and cardiac weakness develop in the very beginning of the disease, as well as those in which severe necrotic angina and an adenophlegmon appear early, *are dangerous*. Such septic scarlet fever is particularly dangerous to children under three years of age.

Mortality depends on the form of scarlet fever and the age and condition of the child. The younger the child the higher the mortality. Before the Second World War mortality averaged 7-10 per cent, while in recent years, in virtue of the milder character of the epidemics and the considerable achievements in scarlet fever therapy (especially because of administration of penicillin), it has decreased almost to zero (it amounts to a small fraction of one per cent). An intercurrent secondary infection (influenza, measles, diphtheria, chickenpox) to the disease always aggravates the prognosis.

Hygiene, care and treatment. Like all other infections, scarlet fever must be treated according to the principle of complex pathogenetic therapy, i.e., by general measures which ensure the patient requisite care, diet and regimen; it must also include methods of etiotropic and stimulating therapy, as well as special treatment of the complications.

Hygienic surroundings are very important for a favourable course of scarlet fever. The room must be *well ventilated* and not overheated. During the warm time of the year the patient may be kept outdoors or in a room with wide-open windows. In winter the room must be thoroughly aired; at this time the patient must be well covered and, if necessary, kept warm with hot-water bottles. The room must not be darkened. Warm (36-38°C) baths are very beneficial.

Care of the skin and the mucous membranes is highly important. Dry and desquamating skin must be greased. To avoid bed-sores, it is necessary in severe cases to place the patient on a rubber ring and to rub down the region of the sacrum daily with diluted alcohol or merely wash it with boiled water.

Older children must (their condition permitting) gargle their *throat and mouth*. The gums, lips and tongue of grave cases and very young children must be carefully cleaned 2-4 times a day with a moist cotton wound on a forceps. A 2 per cent boric acid solution, very weak potassium permanganate solutions, a 0.85 per cent NaCl solution or even boiled water may be used. The lips, tongue and nostrils have to be coated with vaseline or vegetable oil. The nasal passages, if they are obstructed with mucus, must be cleaned with a tampon coated with vegetable oil or vaseline. The eyelids must be cleaned with a cotton soaked in boiled water or a boric acid solution and coated with vaseline.

In severe cases the patients must not be put on a night pot, but a *bedpan* must be placed under them instead.

Diet. During the first stage of scarlet fever, when the patient has a high temperature and swallows with difficulty because of the affected fauces, he must be given liquid or semiliquid food. The diet must consist of fresh and sour milk, tea with cream, cereals, jellies, and broths with cereals and yolks. It is very good to give the patient juices (grape, apple, berry, carrot and tomato juices) in doses of 25, 50 and 100 ml a day, depending on the patient's age. At this time the patient requires adequate nourishment and an abundance of liquids (the patient must often be given to drink, especially in severe cases). The portions must not be large; the patient may be fed 5-6 times a day, very young children—every

two hours (8-10 times a day). The food must be well and palatably prepared and heated.

After the temperature has dropped and the condition of the fauces has improved the patient *may be transferred to an ordinary nourishing diet* with the exception of coarse foods. The child need not, as it was done before, be deprived of meat and be kept on a milk or protein-free diet. The child may be given meat in the form of rissoles and quenelles, fish, chicken, soft-boiled eggs (only the yolks) or hard-boiled eggs (not more than 1 egg a day for older children), meat and vegetable soups, mashed potatoes, puddings, white bread, curds, and butter. The diet must consist of a variety of foods. The child must be given *fresh fruit and vegetables*. *Fish-liver oil* is very beneficial. Coarse foods—raddish and pickles—and tinned foods are not recommended.

This diet must be prescribed only if the work of the kidneys is kept under thorough control and must be correspondingly altered as soon as there is any suspicion of inflammation of the kidneys (see below). If the child has a poor appetite, he must be made to eat the requisite portion.

The diet of grave patients, who run a fever for a long time, have necrosis, are undernourished and have no appetite, presents considerable difficulties and requires serious attention. At the very first symptoms of severe intoxication with clouded consciousness it is recommended to give the patients plenty of liquid (tea with sugar and juices, a 5 per cent glucose solution); the patients may be given enemas of a 5 per cent glucose solution or the glucose (sterile) may be administered subcutaneously. The child must not be allowed to starve and must not be given monotonous, carbohydrate, innutritious food. The food must be easily assimilable and palatable and must contain enough protein (especially milk protein) and nourishing fats. The child must receive fresh fruit and vegetables or juices. If the child is undernourished, he must be given fish-liver oil even if he runs a fever. In cases of high fever which persists at constant figures *warm daily baths* (37°C) are helpful. If the fever is very high (40° and higher) and is attended with clouded consciousness, the patient may be administered once or twice a day cold packs (temperature of the water—36-38°C) for 10-12 minutes each time. A cold fomentation or ice bag is applied to the head.

Septic forms of scarlet fever and its purulent complications are now very successfully treated with penicillin.

Penicillin should be used in all cases of scarlet fever with considerable affections of the fauces and nasopharynx, especially in

very young children and in cases of purulent complications. It is administered intramuscularly in doses of 100,000-200,000 units a day for a period of 7-10 days or more, depending on the clinical indications. In recent years, owing to the milder character of scarlet fever epidemics and extensive use of penicillin, serum is very rarely administered. Administration of scarlet fever antitoxic serum is indicated in severe toxic forms of the disease and as early as possible.

Blood transfusion (50-100-150 ml) is resorted to in severe septic cases, but it is contraindicated in cases of marked toxicosis.

Intramuscular injections of 60 ml of normal human serum and intravenous as well as intramuscular administration of plasm are effective in septic cases and in cases of moderate severity with early complications. After the injections a drop in temperature and an improvement in the general condition and local symptoms are observed. If the injections have failed to produce an adequate effect, they may be repeated within 2-3 days.

Very important for a favourable course of scarlet fever and prevention of complications is a correct system of hospitalisation (simultaneous placement of patients in wards, an extensive aeration regimen and thorough implementation of all measures aimed at preventing cross infection). The fauces and nasopharynx are disinfected with antibiotics, the number of complications is reduced and the period during which scarlet fever convalescents are contagious is shortened, thereby decreasing the hospitalisation period of scarlet fever patients.

Treatment of complications. Moderately severe cases of scarlet fever require no administration of cardiacs.

Cardiacs have to be used in severe cases, especially in toxic scarlet fever with symptoms of cardiac weakness. Subcutaneous injections of caffeine, cordiamine (nikethamide), and camphor, and intravenous administration of 10-20 per cent glucose solution with an addition of ascorbic acid may be recommended.

Purulent complications (nasopharyngitis, necrotic angina, sinusitis, lymphadenitis, arthritis, otitis, mastoiditis, empyema, etc.) are treated with penicillin in adequate doses (200,000-300,000 u per day for a period of 7-10 days, depending on the severity of the manifestations). In cases of a protracted and severe course of these manifestations it is necessary to use a combination of antibiotics (injections of streptomycin at the rate of 25,000 u per 1 kg of weight per day or biomycin (chlortetracycline) per os in a dose of 25,000 u per 1 kg of weight per day administered in four stages). Local treatment is administered at the same time.

Severe processes in the nose and nasopharynx require administration of a 2 per cent protargol solution or adrenalin (1:10,000) in the form of drops.

For *inflammation of the lymph nodes* heat in various forms (hot-water bottles, hot compress, sun lamp) is recommended. Ultraviolet irradiation of the region of the neck with erythematous doses is productive of particularly good results. In cases of dense lymphadenitis (to hasten the suppuration) and in adenophlegmons linseed fomentations may be used. As soon as the nodes begin to suppurate an incision must be made.

Rest and heat are prescribed in cases of *synovitis*.

Purulent arthritis often requires surgical intervention.

In cases of *inflammation of the middle ear* hot compresses are applied to the ear and behind it. As long as there is no perforation warm oil or 5 per cent phenol glycerite may be instilled into the ear.

In cases of clearly defined bulging of the drum (as shown by otoscopy) the latter is punctured (*paracentesis*).

In incipient *mastoiditis* ice and later heat are applied to the ear. This complication requires serious attention; if the process progresses surgical intervention must be resorted to.

Early latent cases of *mastoiditis* are particularly dangerous and must be operated as soon as possible to prevent development of thrombosis of the sinus and sepsis.

The wound must be cared for according to general surgical rules.

Treatment of nephritis. In the treatment of nephritis it is very important carefully to watch the patient's general condition, his complaints, nervous manifestations, cardiac activity, blood pressure, weight, micturition, changes in the urine, and intestinal function. The patient must be confined to bed and kept warm by means of hot-water bottles; he must be ensured rest, prescribed a special diet, and given warm baths (37°C) every other day. In cases of *rapidly increasing edema* and retention of water the patient must be administered *hot packs with oilcloth* or given *hot baths* followed by packs (temperature of the baths—39-40°C).

During the initial period of nephritis the diet must be based on the principle of sparing the kidneys, but starvation must be reduced to the minimum. An exclusively dairy diet is inadvisable.

A diet completely excluding proteins is no longer prescribed in cases of nephritis. Large quantities of salt are harmful because it is difficult for the kidneys to excrete the salt during the acute stage of the disease; in the beginning it is also necessary to limit

the intake of water for the same reason. The amount of liquid is limited, especially in cases with edema and marked hypertension (in determining the permissible quantities of liquid the data furnished by measurements of the excreted urine should be used as a guide). In the first days the patient is given salt-free food containing no animal proteins. So-called "sugar days" (100-400 g of sugar in fruit juice, tea at the rate of 10-12 g per 1 kg of the patient's weight) are recommended in the beginning of the disease.

The following is an approximate diet for nephritis patients:

A starvation diet may be prescribed and 100-150 ml of liquid may be allowed on the first day. On the second day the amount of liquid may be increased to 300 ml and administered in the form of tea or milk; the patient may also be given some jelly, thin gruel, a little white bread and an addition of 100-200 g of sugar a day. On the third day the patient may receive 400-600 ml of liquid, a somewhat larger amount of food and 200 g of sugar.

Then, while the salt intake is still limited, the diet may be extended, i.e., the patient may be given fresh and sour milk, curds, jellies, cereals, fresh and stewed fruit, vegetables, bread, butter and fruit juices. The amounts of liquid may vary with the severity of the case: from 500-700 to 1,000 ml.

In grave cases with edema salt must for some time be completely excluded.

During convalescence, as the excretion of water improves, the patient may be allowed more liquid and given a greater variety of food. Milk, eggs and a moderate amount of meat and fish are not contraindicated.

In mild forms of nephritis small quantities of animal proteins (fresh fish, eggs and boiled meat) may be added to the diet at a rather early date (in the second week of the disease).

Keeping the patient on a salt-free and meatless diet for a long time is inadvisable and unreasonable. In cases of prolonged nephritis the diet must therefore be extended by addition of small amounts of salt and valuable proteins, with due regard for the clinical data and biochemical indices, the level of residual nitrogen in the blood in particular.

No diuretics, except the usual doses of caffeine, may be used.

It is important to keep the bowels open. After the initial stage of nephritis and in cases of diminished hypertension baths (37-38°C) followed by packs are indicated.

In cases of impending uremia sugar days, limitation of liquid and intravenous administration of a 20-25 per cent glucose solution are recommended. Sudorifics (hot packs with oilcloth and

hot baths) must be used cautiously at this time. During attacks of uremia the patient is administered 2 per cent chloral hydrate in an enema, a lumbar puncture, a venesection (100-200 ml) and oxygen. In persistent convulsions the patient is given intramuscular injections of a 25 per cent magnesium sulfate solution in a dose of 0.2 g per 1 kg of the patient's weight. It is necessary to watch the patient's heart, administering, when indicated, cardiazol, camphor, and glucose intravenously.

Scarlet fever patients are usually allowed out of bed after the 21st day of the disease. If a patient has nephritis he may be allowed out of bed after the disappearance of albumin and blood from the urine, relief of hypertension and in the absence of cardiac changes. In mild cases the sick child may be allowed out of bed sooner, provided his urine is kept under systematic control and he shows no cardiac symptoms. In severe cases the confinement to bed is, naturally, prolonged.

Prophylaxis. Prevention of the spread of scarlet fever requires early diagnosis of the disease and early hospitalisation or isolation of the patient at home (the latter with permission of an epidemiologist), disinfection, and implementation of necessary measures with respect to those with whom the patient was in contact.

The following measures are taken:

- children and adults working in children's institutions are quarantined for 12 days and kept under observation (daily examination of the fauces and skin and measurement of the temperature);

- children who have had no scarlet fever are refused admission to children's institutions for 12 days from the time the children they were in contact with were hospitalised; children who had the disease are not admitted until after the terminal disinfection;

- admission of new children to children's institutions in which there were cases of scarlet fever is suspended for 12 days from the moment the last patient was hospitalised; formaldehyde or a combination of formaldehyde with moist disinfection is recommended as a terminal disinfection.

In discharging patients from scarlet fever departments the physician must be guided by the condition of the fauces and nasopharynx, as well as the presence or absence of complications. Good hospital conditions (separation of new patients from convalescents, adequate aeration, highly hygienic regimen in the departments, etc.) play the decisive role in controlling the contagion that may be spread by scarlet fever convalescents. The patient's parents must be given appropriate instructions at the time of the patient's discharge. At least partial separation of children who

have had no scarlet fever from those discharged from hospital should be recommended for not less than two weeks.

In the U.S.S.R. children who have had scarlet fever may be discharged (since 1952) after the 21st day of the disease, provided the rules of hospitalisation envisaged in the instructions were observed. According to these instructions, the departments from which the children will be discharged after 21 days of the disease must consist of separate wards. Three wards are required for this purpose. The department must have an exact number of permanent beds and must not be allowed to become overcrowded.

Two methods of admitting patients to the department may be used: the department may be filled within 1-3 days (the number of incoming patients permitting) or the separate wards may be filled within the same period of time. The patients of the different wards must have no contact with each other. A maximum of attention must be devoted to airing the wards, which requires a special routine, i.e., fixing of definite hours of the day and night for opening the windows or transoms.

The clinical indications for discharge after the 21st day of the disease are as follows: 1) good general condition; 2) absence of complications at the moment of discharge; 3) normal temperature for at least five days prior to discharge; 4) absence of inflammatory symptoms in the fauces and nasopharynx at the moment of discharge (absence of succulence, hyperemia of the tonsils, mucous or purulent discharges from the nose); 5) blood test showing an erythrocyte sedimentation rate of not higher than 18-20 mm and a leukocyte count of not more than 15,000.

All patients discharged from hospitals after scarlet fever must be kept under observation at home (by an epidemiologist, district physician or community nurse and, if need be, by the attending physician) according to a definite scheme.

Artificial immunisation against scarlet fever has not justified itself, for which reason prophylaxis consists mainly of general antiepidemic and sanitary measures.

MEASLES

Etiology. The disease is caused by a virus which is unviable outside the human organism and is quickly destroyed. During the prodromal stage and the first days of eruption the virus is constantly found on the mucous membranes of the respiratory tract, the mouth and conjunctiva, as well as in the patient's blood.

Epidemiology. Man is extremely susceptible to measles. At least 95 per cent of all people contract measles which is thus one of the most widespread diseases. The idea that measles is a disease that affects only children has formed because most people contract it in childhood.

If measles is brought into an area which has not known this disease for a long time and where very few people had had it before, it gives rise to vast epidemics which affect almost all of the population. Such an epidemic on Faroe Islands was described by Panum in 1846.

The disease similarly spreads among children who have not had measles when brought into children's collectives, if no quarantine is established and no specific preventive measures are carried out.

The disease incidence is highest among children under five years of age; measles is rarely contracted by infants under six months of age and only as an exception by breast-fed babies under three months of age, which is due to the immunity acquired by them through the placenta and the maternal milk (passive immunity).

In the very rare cases in which the mother contracts measles shortly before parturition the child is born with measles as a result of intrauterine infection.

Always existing in populated areas measles may from time to time produce extensive epidemics, usually every 2-4 years (periodicity of epidemics). The incidence of measles is characterised by seasonal fluctuations, i.e., it increases during the cold time of the year (late autumn and during the winter and spring months) largely because of the change in the living and hygienic conditions at this time of the year (overcrowding, insufficient use of air and light, etc.).

The measles patient is the only source of infection. The patient becomes contagious from the very beginning of the prodromal stage and remains such to the end of the eruption (at the expiration of five days after the beginning of the eruption the patient is no longer contagious; the period of contagiousness is five days longer if the patient has any complications).

The infection is transferred from patients to healthy people during sneezing and coughing by means of droplets of moisture containing the causative agent.

As a rule, measles is not transmitted through a third person or things with which the patient was in contact; such transmission of the disease is observed extremely rarely and is possible only during direct contact maintained between the patients and healthy

people, by the attending personnel or through passage of infected objects from patients to healthy people.

Carried by currents of air the measles virus can penetrate through cracks in walls and keyholes into adjacent premises and through ventilation pipes to other stories of the building in which there is a measles patient. This accounts for the spread of measles which sometimes takes place without direct contact with measles patients.

Since the measles virus is unviable, no disinfection of the premises is required after the end of the disease or removal of the patient from the premises; it is merely enough to air the premises well and clean them up carefully.

Poor living conditions—overcrowding, uncleanness and failure to observe the rules of hygiene—are conducive to the spread of the disease and an increase in mortality caused by measles.

One attack of measles usually confers lifelong immunity, the organism of the person who has recovered from measles retaining measles antibodies for the rest of his life (the preventive properties of the blood serum of adults are based on this).

Measles recurs very seldom, not more than in 1-1.5 per cent of the cases, and mainly in debilitated children who, in addition to measles, were simultaneously affected with other diseases owing to which they apparently failed to elaborate sufficiently reliable immunity to measles.

According to one view, children who have recovered from a mild form of the disease, so-called mitigated measles, do not always acquire immunity after being administered antimeasles inoculations. However, special observations conducted over a period of years disprove this point of view. But in the practice of administering antimeasles inoculations it is customary, in cases of contact with measles patients, to regard the children who formerly recovered from mitigated measles as not immune and subject to inoculation. This is based on the fact that in diagnosing mitigated measles, which exhibits mild symptoms, mistakes are likely to occur and another disease, simulating measles, may be diagnosed as measles.

Pathogenesis. The measles virus exerts a very complex and peculiar influence on the organism. Allergic manifestations are very clearly defined during the initial stage of the disease, the severity of measles being determined by these manifestations and early complications, rather than by manifestations of a specific toxicosis which is typical of scarlet fever, diphtheria and dysentery and is but feebly marked in measles.

At the height of its development measles produces numerous manifestations characteristic of anaphylactic states and resembling the serum sickness

(character of the eruption, catarrh of the mucous membranes, leukopenia and other blood changes, vagotonic reactions on the part of the vegetative nervous system, etc.).

Measles causes two kinds of disorders in the organism because, on the one hand, the measles virus is a strong poison for the cells of the nervous system and the epithelium of the mucosa and, on the other hand, it exerts a strong allergising influence. These properties of the virus are manifested in the vulnerability of the central nervous system and the epithelium of the mucosa, a considerable reaction of the lymphatic and homopoietic apparatus, intense disorders of the barrier functions, disturbance in the vitamin balance (primarily, as regards vitamins A and C), diminished immunity and increased general and local susceptibility to secondary infection, especially, in the systems of the organism in which the action of the causative agent of measles is clearly pronounced (the respiratory and digestive tracts).

Thus the measles virus paves the way for the appearance of complications of various etiology.

The aforementioned changes in the organism's immunobiological state caused by measles explain the necessity of creating the most favourable atmosphere and regimen for measles patients, especially for very young children.

Clinical findings. There are four clearly defined stages in the course of measles: 1) incubation (latent), 2) prodromal, 3) eruption, and 4) convalescence (during which pigmentation of the eruption is observed). The incubation stage (from the moment of infection to the beginning of clinical manifestations) lasts 8-11 days, seldom up to three weeks.

During this stage there are no clinical changes, and only peculiar morphological and physicochemical changes in the blood are noted (at first an increase and then a drop in the leukocyte count, a change in viscosity, coagulability, etc.).

A prolonged *incubation stage* (21-28 days) is observed after vaccination of children who were in contact with measles patients and in cases in which a patient suffering from another infectious disease (for example, scarlet fever) contracts measles.

The *prodromal stage* lasts 3-5 days, and 12-15 days thus usually elapse between the moment of infection and the appearance of the eruption.

The prodromes begin with a rise in temperature (38-39°), a *change in the general condition and catarrhs of the mucous membranes*. The patient develops coryza, conjunctivitis and a cough (usually dry and persistent and not infrequently harsh and barking, as in diphtheritic croup).

The skin usually remains clear; during the prodromal stage an eruption sometimes appears on the trunk, more seldom on the face, in the form of small scattered spots, much less frequently like that of scarlet fever. The changes in the oral and faucial mucosa



Fig. 47 Filatov's Symptom in Measles



Fig. 48. Eruption in Measles

are the most characteristic. Examination of the fauces and mouth during the prodromal stage reveals a very important symptom first described by Filatov (*Filatov's symptom*). This symptom consists in a fine branny desquamation of the epithelium of the buccal mucosa at the molars, in the beginning often at the second lower molars, as well as on the internal surface of the lips and on the gums in the form of small whitish glistening spots surrounded by a slight redness (Fig. 47). The mucosa loses its lustre, becomes rough and assumes a variegated appearance. These spots usually appear two, sometimes three or four, days before the eruption.

Filatov's spots are sometimes very small and not clearly defined, and their detection therefore requires good lighting. *Spotted angina* is noted in the fauces at the same time or somewhat later.

Filatov's symptom is pathognomonic of measles, i.e., is observed only in this disease, and is very important for it makes it possible to diagnose the disease in the beginning of the prodromal stage, before the appearance of the eruption, to isolate the patient and to administer the prophylactic inoculations which may prevent the development of an epidemic.

Eruption. At the end of the fourth day of the febrile process all the symptoms reach their greatest intensity and a typical maculopapular eruption begins to appear (Fig. 48).

First of all it appears on the face, forehead and behind the ears, and then spreads over the whole face, covering particularly plentifully the middle of it (nose, chin, lips); in this respect the measles eruption radically differs from that of scarlet fever in which the middle of the face remains white (scarlet fever triangle). On the *second day* the eruption extends to the *trunk* and on the *third day*—to the *limbs*.

The measles eruption is often observed to spread over the entire body on the very first day, but even in these cases the eruption seems to occur in stages, i.e., it is more intense on the trunk and limbs on the second and third day.

The eruption is maculopapular, i.e., changes in the skin begin with formation of small papules—spots somewhat elevated above the level of the skin, which then grow larger, assume irregular serrated outlines and, when viewed from the side or touched, noticeably bulge above the level of the surrounding skin (exudative character of the eruption).

In some places the eruption merges in fields of irregular form, but even when it does so over large sections of the skin parts of unaffected skin always remain. The colour of the eruption may differ very widely—from pink to deep red and cyanotic. It also

varies with respect to its abundance. Small hemorrhages not infrequently appear at the same time; however, these hemorrhages are not of negative prognostic significance. The eruption lasts about four days and then begins to fade in the same sequence as it appeared.

Dark spots remain on the site of the eruption. The pigmentation lasts one to one and a half weeks and may be of value in diagnosing measles retrospectively (i.e., in cases in which the patient was not under medical observation at the height of the disease).

The fading of the eruption is sometimes followed by a slight branny desquamation on the face and trunk (unlike the desquamation in scarlet fever).

A rather grave picture is observed at the height of the process (on the second or third day of the eruption): high fever, general condition very much disturbed, face puffy and covered with an eruption, eyelids adhering to each other because of a discharge, rhinitis, intense, dry and persistent cough, fauces swollen and red.

A frequent liquid stool, green-coloured and containing mucus is often observed, especially in very young children (sometimes it occurs as early as the prodromal stage). During the prodromal stage and in the beginning of eruption the symptoms include leukopenia, lower neutrophil count and thrombopenia. Filatov's symptom disappears towards the end or even the middle of the eruption. A moderate, general, succulent enlargement of the peripheral glands is noted. The urine often shows a positive diazoreaction.

Simultaneously with the fading of the eruption (on the fourth or fifth day of it) the patient's general condition begins quite rapidly to improve, the catarrhs passing somewhat more slowly.

The entire febrile process lasts about 7-8 days. However, irritability, poor sleep, lack of appetite and intestinal instability not infrequently persist after the drop in temperature.

Forms of measles. There are various forms of measles differing in severity.

Very mild forms of the disease are sometimes observed; in these cases all the symptoms are very feebly marked, the process passes rapidly and produces no complications. Not infrequently the different stages of measles take an atypical and accelerated course with a slight temperature reaction, mild catarrhal symptoms and eruption. This *mild* course is often observed in infants under 4-5 months of age when the passive native immunity begins to wear off but has not yet completely disappeared. It occurs particularly frequently after administration of antimeasles inoculations in cases in which the disease could not be prevented and has

developed in a mild form (so-called mitigated measles). In these cases incubation is usually prolonged, the prodromal stage is absent or shortened to 1-2 days, the catarrhal symptoms are feebly marked, Filatov's symptom is frequently absent or barely outlined, the eruption is for the most part scanty (although sometimes it is quite abundant but appears rapidly without the development characteristic of this stage of usual measles); the disease runs a very mild general course, as a rule, without complications.

Contrariwise, *severe forms* of the disease are observed in other cases. Their severity is most frequently determined by rapidly developing pneumonia attended with cyanosis and dyspnea.

Hypertoxic forms of measles, when the patient dies from intoxication without complications, are observed much more rarely, almost as an exception. Hemorrhages into the skin and the mucous membranes are sometimes observed in these cases. Usually, however, death from measles is due to complications.

Complications. The most frequent and dangerous complications occur in the respiratory tract (especially, pneumonias). The mucous membranes of the respiratory tract are intensely irritated by the measles toxin which not only destroys the epithelium, but also penetrates deeper and causes inflammation of the tracheal and bronchial walls, the peribronchial tissue and the pulmonary parenchyma.

In connection with this the *laryngitis* and *bronchitis* observed during measles not infrequently run a severe and prolonged course.

Morbillous laryngitis sometimes produces a picture resembling diphtheritic croup. During the prodromal stage it often simulates false croup. Such early laryngitis terminates, for the most part, safely since the changes underlying it are not deep; only in rare cases is it likely to result in stable stenosis.

After beginning during the prodromal stage and almost disappearing towards the pigmentation stage laryngitis sometimes *becomes aggravated again* on the 8th-10th-12th day of the disease, but in some cases only sets in at this time. This aggravation or onset of laryngitis may often be connected with the so-called "second allergic wave" during which the patient's general condition grows worse and a number of complications appear.

Such late laryngitis is more dangerous since, being based on necrotic-ulcerative lesions of the larynx, it either runs a sluggish and prolonged course with aphonia and soundless coughing, sometimes leading to development of purulent laryngeal perichondritis, or takes a rather rapid course with increasingly developing stenosis which requires surgical intervention (in these cases a tracheot-

omy, rather than intubation, is indicated). It should be remembered and constantly borne in mind that development of stenosis in measles may be due to intercurrent diphtheritic infection (combination of measles and diphtheria). A differential diagnosis between morbillous laryngitis and true diphtheritic laryngitis is often very difficult. The negative results of bacteriological examinations necessarily conducted in such cases cannot be of decisive importance. In these cases it is therefore recommended to administer antidiphtheritic serum, using the clinical findings as a guide. The entire acute period of the disease (the eruption stage) is usually attended with tracheobronchitis.

Morbillous pneumonia is caused by the measles virus and an intercurrent secondary diplostreptococcus infection. It may set in as early as the prodromal stage of measles, but more frequently develops during the eruption or later. Early pneumonia is prognostically more dangerous, especially in very young children suffering from rickets and hypotrophy.

Morbillous pneumonia is usually of a diffuse character, more frequently *acinous (lobular)*, and is attended with bronchitis (bronchopneumonia). The process gradually involves large sections of pulmonary tissue and causes wet coughing, dyspnea, and high irregular fever. Such pneumonia not infrequently produces bronchiectasis (dilation of the bronchi) in connection with the aforementioned changes in the bronchial walls (peribronchitis) and shrinking of the lung caused by development of connective tissue (cirrhosis). Pneumonia is not infrequently attended with *purulent pleurisy*. Sometimes *gangrene and pulmonary abscesses* develop.

Pneumonia most frequently affect children under 3 years of age and is the most frequent cause of mortality from measles.

In debilitated children, as well as in cases of late treatment, pneumonia runs a protracted course, lasting for weeks, aggravating dystrophy and adding purulent foci. Modern methods of treating pneumonia are effective very largely because the disease is diagnosed and treated in good time.

Affections of the digestive tract constitute another very frequent complication; various forms of *stomatitis* are not infrequently observed, *gangrenous stomatitis* (so-called *noma*) being the most dangerous form. This complication also occurs in certain other diseases, usually in debilitated, malnourished or undernourished children (as a result of typhoid fever, dysentery). The disease begins with an infiltration of the buccal mucosa, which extends into the interior, involving the skin, and in the centre becomes gangrenous, sometimes the entire cheek and the lips, and often the

jaw becoming necrotic. This complication most frequently terminates lethally, but sometimes the process is arrested and the patient recovers. Today noma occurs very rarely, mainly in emaciated children. In girls such affection may develop in the region of the labia.

In some cases septic cervical lymphadenitis of streptococcal etiology may develop.

Intestinal complications are very frequent. Diarrheas caused by the action of the measles virus are observed during the prodromal stage and during eruption; usually they terminate towards the beginning of the convalescence stage. Later colitis with mucosanguineous diarrhea is often the result of dysenteric infection; it develops in children formerly affected with dysentery or those who were in contact with dysentery patients. Colitis during measles often runs a protracted and stubborn course, especially in very young children; it considerably aggravates the condition, leads to dystrophy, emaciation and not infrequently to death.

In addition to the respiratory and digestive tracts the mucous membranes of other organs are also affected during measles, *conjunctivitis*, *blepharitis* and *otitis*—simple and suppurative—being observed.

Otitis is a complication which not infrequently attends measles. Early morbillous otitis is often catarrhal; compared with scarlet fever it rather rarely leads to perforation of the eardrums or to the necessity of their paracentesis.

Late otitis which develops after eruption runs a more severe course; it is usually suppurative, may take a protracted course, be aggravated by mastoiditis (although much less frequently than in scarlet fever) and serve as the source of sepsis and sometimes pulmonary abscesses.

Such a protracted course of otitis, when the process continues despite early and adequate penicillin therapy and the entire complex of therapeutic measures, requires particular attention and, if mastoiditis develops, timely surgical intervention.

The rare complications of measles include encephalitis which usually develops at the time the eruption fades (on the fifth or sixth day of the disease). In some cases it terminates in complete recovery, but may also lead to permanent neuropsychic defects. Considering the viral etiology of measles and the ability of the virus to affect the nervous system, the specific genesis of this form of encephalitis may be surmised. Purulent meningitis observed during measles is a result of secondary infection in complications of measles produced by pneumonia and otitis.

Measles produces a very characteristic effect on chronic infections, especially tuberculosis.

The tuberculous process which had run a benign course before measles was contracted may, after measles, often become aggravated and assume an active progressive form (miliary tuberculosis). It is therefore necessary particularly strictly to safeguard tuberculous children against infection with measles and in case of any contact immediately to segregate them and administer at the earliest opportunity a maximal dose of human measles immune serum or gamma globulin.

The cause of such sensitivity of the tuberculous process to measles lies, as it is supposed, in the frequency of affection of the respiratory tract and the presence of peribronchitis which involves in the inflammation the bronchial glands containing the encapsulated focus; moreover, measles causes a state of altered reactivity and reduced immunity (the positive Pirquet test becomes negative).

Measles may not only aggravate chronic infections, but, owing to the reduced immunity and lowered resistance, may also facilitate attacks of any other infectious disease, which at the given moment surrounds the patient, or activate the disease which was until then harboured by the patient but produced no clinical manifestations. Thus measles cases are known in which a negative Schick test becomes positive, the titer of the Widal test decreases, dysentery becomes aggravated, scabies and pyoderma are revived, and other infections are added.

These facts are very important with respect to hospitalisation and care of measles patients.

Prognosis and mortality. The prognosis depends on the age and condition of the child, and on whether or not there are any complications. Measles is the most dangerous for children under three years of age, especially for weak children affected with rickets and tuberculosis.

Uncomplicated measles is very seldom likely to terminate in death. Measles patients die of complications, mainly *pneumonia*, while very young children also die of *colitis*, *emaciation sepsis* and *noma*. *Tuberculosis* developing after measles may likewise lead to death.

Owing to the effective results produced by the therapy of morbillous complications in recent years, particularly the administration of sulfa drugs and antibiotics, as well as the extension of the whole complex of therapeutic measures, mortality from measles has sharply decreased.

Hygiene, care and treatment. The considerable changes caused by measles in the immunobiological state of the patient necessitate the creation of the most favourable environment and therapeutic-protective regimen which help to raise the resistance of the patient's organism.

Hygiene is of enormous importance. Special efforts must be exerted to *provide measles patients* with fresh air, for which purpose the *premises must be frequently aired*. In warm weather the patients may be kept outdoors. The temperature in the room must not be high (15-17°C).

Under no circumstances must the premises be darkened. Light is a most important biological factor which enhances the resistance of the organism. Since the patient is to a certain extent affected with photophobia he must be placed so that the light does not fall on his eyes. The *light must not be shaded with red* and there must be no red light in the room since this does not help the process and only irritates the patient.

The patient must *bathe* regularly and wash *every day*. Depriving the patient of water is very harmful.

People harbour some of the most harmful prejudices as regards measles; for fear lest the child "catch cold" during measles the child is deprived of fresh air without which he cannot properly recover; for the same reason the child is not bathed or washed; the windows are shaded with dark or red curtains. All this possibly accounts for the severe course of measles, its numerous grave complications and high mortality observed among the children of the families in which these prejudices still persist.

The measles patient must be given nourishing food containing adequate proteins, fats, carbohydrates and vitamins. During the initial stage of the disease, when the children, particularly very young children, have a liquid stool, the food must be liquid or semiliquid and easily assimilable—milk, especially varieties of sour milk, kissel and stewed fruit. Later meat, fish, vegetables and fruit may be added.

Of course, nursing is enormously important for infants in arms.

During the convalescence stage the patient receives fish-liver oil; vitamins in the form of juices and preparations must be given during all stages of the disease and upon recovery (it should be remembered that during measles the organism becomes particularly impoverished in vitamins A and C).

The *mucous membranes and skin require thorough care*. The eyes are washed every day (twice a day and more) with a 2 per cent boric acid solution or boiled water and the eyelids are coated with vaseline.

In cases of acute conjunctivitis and photophobia, especially in children with impaired conjunctiva and cornea (*phlyctena*, *keratitis*), which are easily aggravated during measles, in addition to washing the eyes with a 0.85 per cent NaCl solution a 2 per cent xeroform ointment may be applied to the conjunctiva.

In cases of copious *discharges from the nose* warm oil is instilled into the nose and, if the patient finds it very difficult to breathe through the nose, an adrenalin solution is instilled into the nose once or twice a day:

Rp. Sol. Adrenalini hydrochlorici 1 : 1,000 1.0

Aq. destill. 9.0

MDS. 2 drops into the nose b.i.d. or t.i.d.

Medication. In uncomplicated measles there is no need for cardiacs because in this disease the cardiovascular apparatus is generally scarcely affected.

Such indications may appear only in very rare cases of severe uncomplicated measles of a toxic or hemorrhagic character. Caffeine, camphor and cordiamine (nickethamide) are administered subcutaneously, and Ringer's solution with glucose—subcutaneously or intravenously.

In diarrhea and in cases of considerable loss of water, when the tissues become less turgid, it is necessary (in addition to administering enough liquid per os) to *administer liquid subcutaneously*. Patients are given, depending on their age, 100-150 ml of *Ringer's solution* with an equal amount of a 5 per cent *glucose* solution.

The symptoms of *tracheobronchitis* are mitigated by administration of hot baths (38-40°C). Per os it is enough to give the patients *alkaline* waters (borzhomi, etc.) or a teaspoonful (dessert-spoonful, tablespoonful) of a 2 per cent sodium bicarbonate solution in hot milk.

During the initial stage of measles *laxatives* should be avoided. Laxatives are generally not indicated in the beginning of *infectious diseases*, whereas in measles they are definitely harmful because they may provoke protracted and stubborn diarrhea, irritating the mucosa already irritated by the measles toxin.

Treatment of complications. The phenomena of *laryngitis*, which threaten development of stenosis, require, in addition to the aforementioned measures of care, hot general and foot baths, mustard packs and peroral administration of bromides in cases of restlessness, i.e., the measures recommended for *diphtheritic croup*.

Since the measles patient may become infected with diphtheria, it is necessary to make sure that the "late" morbillous croups are not of diphtheritic etiology (repeated seroculture) and, upon the slightest suspicion, to isolate the patient, remove him from the measles department and administer antidiphtheric serum (10,000-20,000 u, repeatedly, if indicated).

Pneumonia is treated according to the general rules; the treatment is based on thoroughly organised care, airing of the premises, hot baths, mustard plasters and mustard packs. A good effect is produced by diathermy and, in protracted forms, pleuropneumonia and pneumonia with abscesses—by treatment with ultra-short waves.

The use of sulfa drugs and antibiotics has ushered in a new era in the treatment of morbillous pneumonias. Administered in the beginning of pneumonia sulfa drugs in a number of cases quickly terminate the process, the temperature drops critically and the child's condition improves. Even in far advanced cases with vast microfocal affections the process is soon observed to terminate. In morbillous pneumonias the medicine is administered in usual doses in milk, fruit juice or kissel. Nausea, vomiting, leukopenia and neutropenia are the side effects observed. Cases with marked side effects necessitate temporary suspension of the treatment and administration of sufficient quantities of water.

If treatment with sulfa drugs fails and if manifestations of a septic type (pneumonia with abscesses, purulent pleurisy, cervical lymphadenitis, perichondritis, etc.) appear, penicillin administered by the usual method proves very effective. In graver cases of pneumonia in little children it is generally preferable to administer penicillin (with or without sulfa drugs) from the very outset.

Suppurative otitis requires timely treatment with penicillin. Local therapy for otitis consists in application of heat, ultrashort waves, and toilet of the ear in cases of pyorrhea.

Treatment of *gangrenous stomatitis* includes gargling and syringing with a weak potassium permanganate solution. In cases of halitosis it is necessary to rub iodoform into the gums and decomposing tissues of the jaw, to administer blood transfusions, anti-gangrenous serum (15,000-20,000 u several times a day) and ultrashort wave treatment.

If the smear contains Vincent's flora, salvarsan solutions (10-15 per cent) are administered locally.

In cases of *colitis* the treatment consists in applications of heat to the abdomen, hot baths, diet as in dysentery, diathermy, ozoce-

rite, chemotherapeutic agents and antibiotics used in the treatment of dysentery (see *Dysentery*).

The treatment for *conjunctivitis*, if the latter takes a graver course and is attended with ulcerous affection of the cornea, consists in application of a 2 per cent xeroform ointment, instillation of cocaine with atropine and penicillin drops into the conjunctival sac. Complications of the eyes must be treated in accordance with instructions of an eye specialist.

Prophylaxis. Measles is very contagious; there is no immunity if the child never had measles before or was not inoculated against measles after contact with a measles patient; the danger of infection in the life of children is therefore very great and is fraught with particularly grave consequences for very young and weak children.

Very strict prophylactic measures must be taken to safeguard children, especially children's collectives (nurseries, kindergartens, hospitals) against measles.

The prophylaxis of measles is effected mainly by antiepidemic measures and passive immunisation.

Early diagnosis of measles and isolation of the patient are of prime importance for timely specific prophylaxis and for expediting the quarantine measures.

A child isolated from other children during the first days of the prodromal stage may not be a source of spreading the disease. Hence, the necessity for the most careful supervision of the children coming or being brought to the collective, especially in the beginning of some febrile disease. Every feverish child suspected of measles must be thoroughly examined (fauces, mucous membranes) and isolated from the other children.

Inoculated children who have had contact with a measles patient are quarantined for 17 days, uninoculated—for 21 days. If the child retains contact with a measles patient the quarantine is correspondingly increased by five days.

In the absence of complications measles patients are discharged from hospital 8-10 days after appearance of the eruption.

The premises are disinfected by airing, washing and moist cleaning; no formalin disinfection is required. The linens are boiled, the clothing is cleaned and aired.

Specific prophylaxis. Artificial passive immunisation is effected, i.e., the healthy children who were in contact with measles patients during the contagious stages (prodromal stage, eruption) are administered subcutaneously the blood serum of a person (child or adult) who recently survived an attack of measles, the serum

containing antibodies which prevent the development of the measles virus in the organism of the infected person.

This method has now been modified, i.e., the *serum of an adult* is administered since nearly all adults had measles in childhood and have acquired permanent immunity to the disease.

The child who was in contact with a measles patient is administered 30-60 ml of this serum intramuscularly or subcutaneously (usually the anterolateral surface of the thigh).

The dose of the serum depends on:

the time of administration—the later after the contact with the patient it is administered, the larger the dose;

the state of the child's health and his age.

The sooner the antimeasles serum is administered, the more reliable the prophylactic effect.

It is usually recommended that the serum should be injected not later than the fourth day after contact (15 per cent of the children contract the disease when the serum is injected on the fourth day after contact with a measles patient, 29 per cent—on the fifth day, 65 per cent—on the sixth day, and 89 per cent—after the sixth day). When small doses of the serum (30 ml) are administered during the first incubation days, the disease is either prevented or considerably mitigated (mitigated measles).

By early administration (before the fourth or fifth day following contact with a measles patient) of large doses (60 ml) of the serum it is possible almost completely to prevent the disease.

Inoculation after the seventh or eighth day of the incubation period is of little avail; despite large doses of the serum it is impossible not only to prevent the disease, but even to avoid a moderate and at times severe forms of measles.

Insusceptibility to measles in inoculated children lasts 3-4 weeks; if a possibility of new contact with a measles patient arises in 3-4 weeks, it is necessary additionally to administer a lesser amount of serum, but not less than 30 ml.

Subject to compulsory antimeasles inoculations are: 1) children from three months to four years of age who were in contact with measles patients; in kindergartens 4-7-year-old children are also subject to inoculation; 2) children in children's hospitals and sanatoriums, regardless of age; 3) children of preschool age (past four) affected with any chronic disease or convalescing from acute diseases; 4) tuberculous children.

In hospital all children are administered at least 60 ml of the serum. In nurseries, in cases of an outbreak of measles, healthy children are administered 30 ml of serum, weak children from

families in which there are tuberculous patients—60 ml to prevent them, if possible, from contracting measles.

In the U.S.S.R. antimeasles inoculations are very widely practised and the methods of preparing serums have been considerably improved. A preparation of gamma globulin has of late been produced from the serum; the concentration of antibodies in this preparation is 25 times that of the initial serum. This makes it possible to administer 1.5-3 ml of gamma globulin containing 20 per cent protein instead of the 30-60 ml of serum; if the gamma globulin contains 10 per cent protein, a dose of 3-6 ml is administered.

Methods of active immunisation to measles are now being successfully elaborated in Soviet research institutes.

GERMAN MEASLES

Etiology. The causative agent is unknown. In its properties it closely resembles that of measles; it is very volatile, unstable in the external environment, and apparently enters the organism in droplets through the respiratory tract.

Epidemiology. As in measles, the infection comes from patients and is not transmitted through things or third persons. The disease is less contagious than measles.

Children between six months and 10-12 years of age are susceptible to infection.

The patient is the most contagious in the beginning of eruption; after the eruption has faded the contagiousness diminishes and completely disappears, as in measles, towards the seventh day of the disease. An attack of the disease confers permanent immunity.

Clinical findings. The incubation period is 10-21 days during which there are no clinical manifestations. Unlike measles, this disease either has no prodromal stage or the latter is extremely feebly pronounced.

The eruption stage usually begins with a rise in temperature which is rarely very high (usually 37.5-38°).

Only in rare cases is high fever (39-40°) observed several hours before the eruption.

The eruption resembles that of measles, but is finer, a paler pinkish-red, less elevated above the skin, and frequently macular rather than maculopapular; its elements are evenly distributed and are of about equal size, for the most part of round or oval form, and not crescentically arranged as in measles.

Usually beginning from the head (behind the ears, on the cheeks and the hairy part of the head) the eruption very quickly (and not in three days as in measles) spreads all over the body; it is most noticeable on the back, the extensor and lateral surfaces of the limbs, and on the buttocks; in most cases the face is not distinctly affected.

There is a weak catarrh of the mucous membranes, a mild conjunctivitis and coryza. The fauces and the mucosa of the cheeks are not infrequently observed to be slightly hyperemic and parti-coloured. Filatov's symptom is absent. There is a characteristic succulent enlargement of the lymph nodes. The *posterior cervical and occipital* lymph nodes become particularly clearly enlarged; their enlargement is often discernible even before the appearance of the eruption and usually disappears several days after the eruption has faded.

The internal organs suffer no particular changes. The blood shows leukopenia (3,000-4,000 leukocytes) with neutropenia and an increase in plasma cells, sometimes up to 15-30 per cent.

The urine is unaffected.

The disease usually runs a mild course. The eruption lasts 2-3 days and disappears without pigmentation or desquamation. Complications are rare. The disease is not dangerous, the prognosis is favourable. The danger lies in confusing German measles with other diseases, especially with measles and sometimes with the serum sickness.

Prophylaxis. Patients may be allowed to associate with children seven days after the eruption. The quarantine for those who were in contact with patients is three weeks. In doubtful cases in which the possibility of measles cannot be completely excluded the patient must be isolated and the children who were in contact with the patient must be given preventive antimeasles inoculations.

Treatment. General hygienic measures, usual diet, baths.

RUBELLA SCARLATINOSA

Rubella scarlatinosa was singled out as a separate disease entity by N. Filatov as early as 1885. Fifteen years later it was described by C. Dukes, an English author. By its clinical picture and eruption this disease resembles scarlet fever, but is a separate, very mild and not dangerous entity. An analogy can be drawn in this respect with German measles which, although very much like measles, is also a separate, very mild and not dangerous disease entity. It is for this reason that Filatov named this disease "rubella scarlatinosa".

The causative agent is unknown. Only the patient himself is contagious. The infection is apparently not transmitted through things or third persons. The incubation period is 15-21 days.

The disease has no prodromal stage. It begins with an abrupt rise in temperature, usually not very high, and a rapid appearance of a punctate scarlatiniform eruption which within a few hours covers the whole body. Mild angina, always of a catarrhal character, is observed. The tonsils are not enlarged. The general condition of the patient is unaffected. The slightly elevated temperature lasts no more than 2-3 days. No complications or desquamation are noted. Mild cases of scarlet fever are not infrequently mistaken for rubella scarlatinosa, which is, of course, unwarranted. It should be remembered that rubella scarlatinosa is a very rare disease and that most frequently there is a mild form of scarlet fever with respect to which all the usual strict prophylactic measures must be carried out.

If the angina assumes a necrotic character, lymphadenitis appears and cardiovascular disorders or desquamation develop, such cases must definitely be regarded as scarlet fever. The disease requires no special treatment except observance of the usual rules of hygiene and keeping the patient for several days on a lighter diet.

Prophylaxis. The danger lies in the possibility of confusing this disease with scarlet fever, for which reason all children suspected of having scarlet fever must be isolated and kept under observation.

It should be noted that the existence of rubella scarlatinosa as a separate disease entity has of late been subjected to doubt. Many physicians hold that most cases of rubella scarlatinosa are mild forms of scarlet fever, while few cases are atypical forms of German measles with a fine eruption. This leads to an important epidemiological conclusion of the necessity of isolating sick children and carrying out corresponding sanitary measures.

ERYTHEMA NODOSUM

Etiology. The etiology of this disease has not been established. Some authors consider it a separate infectious disease entity, others closely associate it with tuberculosis or even regard it as a peculiar tuberculous manifestation.

Some authors ascribe this disease to manifestations of rheumatism. It is more correct, however, to consider it an entirely separate acute infectious disease entity.

Epidemiology. The disease most frequently occurs in childhood; it affects children between three and ten years of age and seldom infants; it is observed in autumn and spring. Minor epidemic outbreaks and family attacks have been described.

Clinical findings. The incubation period is usually 2-3 weeks.

The disease may begin quite acutely with high temperature and symptoms of general intoxication (headache, vomiting, sometimes clouded consciousness); these symptoms are soon followed (within 2-3 days) by *characteristic eruptions* (sometimes these

eruptions appear simultaneously with the aforesaid symptoms). In other cases this (prodromal) stage lasts 7-18 days and produces a temperature curve which resembles that of typhoid fever.

The eruptions are very characteristic, appearing as quite sizable *bluish-brown enlargements* (nodes) on the *anterior surfaces of the shanks*. The nodes are *hot to touch and painful*. They also often appear on other parts of the body—forearms, thighs, and even face, in which case they are smaller and more rose-coloured.

The nodes appear in several stages, each stage attended with a rise in temperature. Within one and a half to two weeks, sometimes sooner, the temperature drops and the nodes fade leaving a pigmentation and a slight desquamation.

Bronchitis, a coated tongue and an enlarged spleen are not infrequently observed during the acute stage; as the temperature drops, the pulse slows down, there is arrhythmia and a slight dilatation of the heart, sometimes attended with a faint murmur; in some cases intestinal disorders are observed. The patient's general condition is usually barely affected and the disease terminates in recovery. Complications are rare and consist in swelling of the joints, pneumonia, otitis and nephritis.

Roentgenological examination during the acute stage, and sometimes soon afterwards, reveals *peculiar changes at the root of the lung* in the form of *infiltrates* simulating specific tuberculous *radicular* infiltrative processes. In erythema nodosum they are usually resorbed, although sometimes they leave indurations.

During the acute stage the *Pirquet reaction* is *clearly positive*, but its intensity quickly diminishes by the time the eruption has faded.

A *fast erythrocyte sedimentation rate* is characteristic of the acute stage (50-80 mm per hour).

Some authors have found the *tubercle bacillus* in the blood, *lavage waters* and *biopsy specimens* taken from the nodes. This has warranted a number of authors (Poncet et al) to consider erythema nodosum a form of tuberculosis.

However, this view cannot be taken for granted; in most cases the course of the disease simulates an *acute infection which confers absolute immunity*. The seasonal character and epidemic outbreaks of the disease, as well as the cyclic nature of its course and the vivid peculiarities of its clinical manifestations with the typical localisation of the affections, also contradict this view.

It may be assumed that this infectious disease possesses a marked *ability to activate the latent* (concealed) *tuberculous process*.

Treatment. During the acute stage the treatment consists in confinement to bed, easily assimilable but nourishing food and observance of the rules of hygiene. In cases of pains in the legs—hot-water bottles or compresses; no medication is required.

Prophylaxis. Since, as it was already stated, the disease in a considerable number of cases *aggravates the tuberculous process* and may give rise to an

infiltrative pulmonary affection, sometimes even a destructive (cavernous) or miliary process, children affected with erythema nodosum should be registered and kept under prolonged observation as *tuberculous cases* (clinic for tuberculous patients); their brothers and sisters must also be examined. Children with the foregoing symptoms of tuberculosis must be placed in the most favourable conditions (sanatorium-type camp, forest school).

CHICKENPOX (VARICELLA)

Etiology. The disease is caused by a very volatile filtrable virus which is found in the patient's blood and in the contents of the vesicles during the outbreak of the eruption.

Chickenpox is a separate disease entity and is in no way connected with either smallpox or vaccinia. Neither of these diseases prevents chickenpox and vice versa.

Data have now been obtained attesting the identity of the causative agent of chickenpox with that of herpes zoster. There are also epidemiological observations indicating a certain relationship between these diseases.

Epidemiology. The disease occurs in sporadic cases and rarely in considerable outbreaks. However, brought into a large group of very young children (nursery, hospital) it causes outbreaks almost as significant as those of measles. Children of the first four years of life are the most susceptible; those past 9-12 years of age are affected less frequently, while infants in the first months of life contract the disease very rarely.

Chickenpox is very contagious, but the contagion is transferred only directly by the patient to the healthy child (air-borne or droplet infection); the disease is not transmitted through things or third persons. The affection is contagious from the very first hour of the eruption (and possibly even earlier), for which reason it is so difficult to control its spread.

Many believe that during the period of crust formation the patient is no longer contagious; it is probably more correct, however, that the patient ceases to be contagious only after the crusts have fallen off.

The causative agent is not viable and easily perishes outside the organism, for which reason the premises and things do not need particularly strict disinfection. As a rule, an attack of chickenpox confers lifelong immunity.

Clinical findings. The incubation period is 14-21 days, most frequently 17 days; during this time there are no clinical manifestations.



Fig 49. Chickenpox



Fig. 50. Faucial Diphtheria

The onset of the disease varies. Prodromal symptoms—a moderate rise in temperature, indisposition, sluggishness, restlessness, infrequently (for the most part in infants) diarrhea and vomiting—are sometimes observed for a period of one or two days after which the characteristic eruption appears.

Most frequently, however, with a moderate rise in temperature an eruption appears simultaneously and at once on different parts of the body; the eruption at first looks like scattered macula from a pinhead to a lentile in size, the macula very rapidly developing into vesicles with a transparent content. The vesicles are located on a barely affected skin, only a small zone of moderate redness surrounding each vesicle.

The chickenpox vesicles differ from the smallpox pustules which at first appear against a pinkish background as papules and then develop into vesicles based on an inflamed red swollen skin. The centre of such vesicles (pocks) is pitted, which is not the case in chickenpox.

In chickenpox the vesicles appear all over the body, most frequently erupting on the hairy part of the head (whence the eruption often begins) and the face, the palms and soles, mucous membranes (conjunctiva, fauces, cheeks, tongue, genitals, external auditory meatus, pharynx and larynx); they soon burst and ulcerate causing difficulties in eating and swallowing, and sometimes produce symptoms of laryngitis. At first the vesicles have a transparent and then a turbid content (Fig. 49).

The eruption occurs in several stages, intermittently, so that the picture of the eruption varies very greatly (is polymorphous).

A macula, nodule, fresh, burst and drying vesicles, and crusts can be seen on any part of the skin; the elements of the eruption are of different sizes.

In smallpox there is no such polymorphism of the eruption which is always uniform on any part of the skin.

In smallpox the eruption breaks out first on the face, mainly on the forehead, the hairy part of the head and almost simultaneously on the back of the hands and fingers; on the second day it appears on the trunk and more densely on the face, and on the third day on the limbs.

The eruption is most abundant on the face and then on the back of the hands; it is less plentiful on the back and very sparse on the internal surface of the thighs and, especially on the abdomen which is almost free of it.

Diagnostically this successive (in stages) spread of the eruption is very characteristic of smallpox. In virtue of this, papules

and vesicles or vesicles and pustules, but never papules and pustules, can be seen on the body simultaneously.

In chickenpox the vesicles burst and dry up in about two or three days and form a crust which falls off within one to two and a half weeks, leaving, unlike smallpox, no scars. Only in rare cases under the influence of secondary purulent infection does a deeper process develop and a small scar form.

The eruption *varies in intensity* from separate vesicles to dense, merging groups. The eruption and drying of the vesicles is attended with itching which makes the patient scratch and tear off the vesicles, thus sometimes leading to secondary purulent infection (abscesses, erysipelas, cutaneous diphtheria).

The *general manifestations* during the eruption are slight: the temperature rises moderately, usually at each new outbreak; it is never high and lasts 5-7 and sometimes up to 10 days; cases with normal temperature are not very rare. Moderate headaches and jadedness are observed. No changes in the internal organs are discovered, the glands become slightly enlarged (if there is no secondary infection). In uncomplicated cases the blood shows leukopenia due to a decrease in neutrophils.

In some cases the eruption of vesicles is attended (sometimes preceded by 10-12 hours) with a rash, sometimes urticarial, sometimes morbilliform, but most frequently punctate, scarlatiniform. In the latter case scarlet fever may often be suspected. In 5-10 per cent of the cases this is observed in infants.

In differential diagnosis it is necessary to remember the ephemerality of the eruption (it usually lasts from a few to 24 hours), its arrangement in crops, the absence of marked angina, not infrequently the presence of vesicles on the oral and faucial mucosa, which precede their outbreak on the body.

At the same time neither angina nor necrosis is observed in the fauces, and there is no desquamation; this distinguishes it from scarlatiniform eruption.

Complications are rare, and in older children the disease constitutes no danger, but in small, weak and emaciated children it may produce severe affections of the skin, namely, abscesses, phlegmons and erysipelas. Such children may develop a gangrenous form of chickenpox with deep disintegration of round ulcers which penetrate to the muscular layer and may lead to *sepsis and death*.

The complications include *nephritis, pneumonia and encephalitis*. Added to other diseases (for example, scarlet fever, measles,

dysentery, diphtheria) chickenpox may noticeably aggravate their course and in such cases run a graver course itself.

Treatment and care. During the period of pyrexia and eruption--confinement to bed. It is necessary to watch particularly carefully that the patient should have *clean* bed-clothes and underwear, and that he should not scratch or tear off the scabs. Moderately warm baths (35-37°C) are recommended during all stages of the process.

If the case involves an affection of the mucous membranes, the diet must be liquid or semiliquid.

The skin is anointed with some boiled vegetable oil, while individual vesicles may be painted with iodine, a strong potassium permanganate solution, a 2 per cent alcohol solution of Loeffler's alkaline methylene blue or brilliant green. It is necessary to prescribe *gargling of the fauces* with a 2 per cent boric acid solution. In septic cases penicillin, streptocide and albucid (sulfanil acetamide) are administered in usual doses.

Prophylaxis. Discharge from the department after *deciduation of the crusts*; quarantine of three weeks for those who were in contact with a patient.

It is now recommended, on the appearance of a chickenpox case, to irradiate with a quartz lamp the entire ward for 2-3 hours every day, having the children wear dark goggles; very few children contracted the disease after this measure had been carried out.

Artificial immunisation. It has been proposed, as a preventive measure, to administer subcutaneously to children who were in contact with chickenpox patients 30-60 ml of adult serum or 3-6 g of gamma globulin. This method may be used with weak children in danger of infection. It is not always possible to ward off the contagion, but the disease, if contracted, runs a milder course.

VACCINATION

The *method of vaccination*, i.e., inoculation of man with *vaccinia* (cowpox), devised by the British physician Edward Jenner in 1796, has played a decisive role in the control of smallpox.

Jenner devised this method on the basis of the following observation: smallpox which raged in Britain at that time was not contracted by women who milked cows affected with a peculiar disease characterised by vesicopustular lesions on the teats and udder; while milking these cows the women contracted this disease but did not contract smallpox.

Jenner artificially inoculated a child with material taken from the vesicles of an infected cow and found that subsequent inoculation of the child with smallpox pus failed to infect the child with smallpox.

Vaccination is inoculation of man with the virus of vaccinia, a very mild cow's disease, which makes man completely immune to smallpox; and, although this disease (vaccinia) is a general process, as a rule, it runs a benign course and very rarely produces complications.

The immunity produced by such vaccination lasts 7-10 years (sometimes less), but then weakens and disappears, thus necessitating *revaccination*.

By order of the U.S.S.R. Ministry of Public Health children must be vaccinated in the 9th or 10th month of life; earlier vaccination may be performed only on epidemic indications (epidemic smallpox cases).

The vaccine used today is a *bovine vaccine* obtained by infecting a calf with attenuated smallpox virus. After the development of smallpox vesicles at the points of inoculation the contents of the vesicles are scraped off with a sharp curet; the detritus is triturated with glycerin and left for a few weeks out in the cold, which kills the microbes contained in the pus but very well preserves the virus. After bacteriological control and a check-up of the activity of the detritus the latter is bottled or ampuled and dispensed for use.

The detritus must be kept in a cool and dark place (at a temperature of 2-6°C). The period of storage is 3-6 months. At higher temperatures the detritus soon becomes inactive.

Vaccination techniques. All rules of *asepsis* must be observed during vaccination. The vaccinator's hands must be thoroughly scrubbed and wiped with alcohol; the vaccination instruments (scalpel, lancet, special steel pen) must be sterilised by boiling.

It is desirable that the child should be bathed and given clean underwear on the eve of the vaccination.

The point of inoculation (usually the lateral surface of the arm on the border between its upper and middle thirds) is thoroughly cleansed with soapy water and wiped with *alcohol* or, preferably, with *ether*; three drops of the detritus—2-3 cm apart (usually two above and one below)—are applied to the dried skin and an *abrasion* connecting the three points is made on the skin (the wound must not bleed, the abrasion being barely stained with blood).

The arm is left uncovered for 5-10 minutes until the detritus has soaked in; no bandages are applied.

Before opening, the ampule or tube with the detritus should be wiped with alcohol or ether.

The child may be washed and bathed until the appearance of an inflammatory reaction (during the first two or three days after vaccination), but, once inflammation and suppuration have begun, the vaccinated arm must not be washed, although the child may be bathed with this arm raised to avoid wetting it.

Upon development of a clearly defined pustule the arm may be bandaged, the dressing containing sterile oil or vaseline.

Clinical aspects of vaccinia. During the first two or three days there are no changes on the skin except the slight reaction to the incision.

On the 3rd or 4th day the abrasion begins to redden and swell, forming a gradually enlarging papule surrounded by a red areola.

Towards the 7th or 8th day the papule develops into an umbilicated vesicle with at first transparent and then turbid contents.

On the 8th or 9th day the reaction is at its height, a redness developing around the pock and the skin feeling hot to touch.

The axillary lymph nodes may become enlarged.

Retrogression of the pocks begins on the 10th or 11th day. The vesicles begin to turn yellow and dry; a yellow-brown crust is formed, the crust falling off in the 3rd or 4th week and leaving the characteristic scar.

Beginning with the 5th or 6th day the entire process is attended with fever (38-39°), itching and general indisposition.

The child is *examined for control purposes* on the 7th or 8th day. In the absence of a reaction the vaccination is repeated for the second time.

Evaluation of the results of vaccination. If the results of the first vaccination are positive, there must be a well-developed papule or vesicle on the 7th or 8th day. The development of even one papule suffices to confer immunity.

Revaccination and evaluation of its results. The immunity conferred by vaccination becomes complete at the time of crust formation. Revaccination must be performed at the age of four, eight, twelve and eighteen years. In cases of revaccination the reaction sets in more rapidly; besides, it is weaker and soon terminates.

Vaccination is considered positive if, on control examination day (3rd or 4th day in cases of revaccination) there is a clearly marked *nodule, vesicle or crust*. The results of the examination are recorded.

If the results are *negative*, the *revaccination is repeated within a month* and, if it fails this time—within another year.

Should the results of revaccination prove negative in all cases, the quality of the detritus used must be ascertained.

According to the instructions for smallpox vaccination (see *Supplement*), should the disease be brought into a community, the entire population must be immediately revaccinated regardless of the date of the previous vaccination.

Complications. Some of the complications are due to secondary infection caused by failure to observe the rules of asepsis during the inoculation, or to subsequent contamination (*erysipelas, purulent lymphadenitis, phlegmon, sepsis, infrequently diphtheria, syphilis, tetanus, pustular eruption*), others to the action of the virus itself. These complications include vaccinal *erythemas* and *secondary pocks* which appear on the adjacent parts of the skin; infection of other parts of the body, *of the eye, genitalia* and skin in cases of eczema in young children.* The disease may thus be transferred, through failure to observe the rules of caring for the patients, from a vaccinated to an unvaccinated child. In cases of *vaccinia* it is therefore necessary faithfully to observe all rules of hygiene and care of the vaccinated child, i.e., to wash the child's hands more often, trim the child's nails, and prevent scratching and contamination of the pocks (clean underwear). In cases of acute reactions the child's arm must be bandaged, the *dressing containing sterile oil or vaseline to protect the pocks*.

In addition to the afore-described complications, primary vaccination is followed—very rarely, to be sure—by postvaccinal encephalitis whose etiology and pathogenesis are not quite clear.

DIPHTHERIA

Etiology. The causative agent of diphtheria discovered by Löffler in 1884 is a straight or slightly curved bacillus the length of the tubercle bacillus but almost twice as thick. Stained with an alkaline solution of methylene blue, especially by the double staining method, it shows peculiar granules (Babes-Ernst bodies) on its ends; these granules are not infrequently seen also in the bacillary body. The bacillus is nonmotile, Gram-positive and aerobic; it develops more weakly under anaerobic conditions. Its best culture medium

* Children affected with exudative diathesis are therefore vaccinated only after disappearance of eczema.

is a coagulated horse or ox serum, its optimum growth occurring at 37°C. To improve its growth, an addition of a small amount of tellurium salts is now recommended; with this method the colonies appear as black grains.

In smears taken from a culture the diphtheria bacilli are arranged very typically, i. e., usually at an angle to each other and seldom parallelly; often they are so intertwined as to look like felt. The diphtheria bacillus produces a very poisonous toxin, virulent to both man and animals. Its virulence (toxicity) is usually tested on guinea pigs. Löffler's bacillus is called the true diphtheria bacillus, as distinguished from the pseudodiphtheritic and so-called diphtheroid bacilli which to a certain extent resemble Löffler's bacillus but are arranged differently, do not produce granularity and, what is most important, elaborate no toxin and are generally nonvirulent.

The existence of three types of diphtheria bacilli has been established in the last 20 years; these bacilli differing in the forms of their cultures and their biological properties are known as the *typus gravis* most frequently found in patients with severe forms of diphtheria, *typus mitis*, usually found in mild cases, and the *typus intermedius*—the intermediate type. Although certain authorities recognise the value of the types of diphtheria bacilli in the clinic and epidemiology of diphtheria, this question cannot be considered settled. What is incontestable is that the severity of the form and the character of the course of the disease (as, incidentally, of any infection) are determined primarily *not by the type of microbe but by the state of the organism*, the peculiarities and reactivity of its nervous system and the influence of the factors in its environment.

Epidemiology. Diphtheric affection occurs as sporadic cases which are likely to produce epidemics. In large cities cases of diphtheria are encountered mainly among the population living under overcrowded and unhygienic conditions. In the past epidemics of diphtheria broke out periodically (every 10-15 years). Increased morbidity is usually attended with epidemics of greater severity, which is attested by the so-called coefficient of severity (i.e., the percentage of severe cases in the total number of diphtheria patients).

A steady decline in diphtheric morbidity has been observed in the U.S.S.R. in the last 30 years.

The active antidiphtheritic immunisation systematically and widely practised in the U.S.S.R. has played an important part in the control of this disease.

Diphtheria is largely a *children's disease* affecting mainly children from one to ten years of age.

The highest disease rate is observed between eighteen months and five years of age. After the age of five susceptibility to diphtheria steadily diminishes. It should be remembered, however, that the disease may affect persons of any age, including newborn and elderly people.

Outbreaks of the disease most frequently occur during the autumn and winter months.

The diphtheria bacillus gains entrance into the human organism in various ways—through direct contact of a healthy person with a patient, through so-called *droplet infection*, sneezing, coughing and talking during which the droplets of sprayed moisture containing the causative agent of diphtheria may be transferred from the patient to the healthy person, through various things, toys, necessities and foodstuffs (milk, etc.), and through contact of a healthy child with a *carrier of bacilli*.

The diphtheria bacillus can live for quite a long time in any medium; it very well tolerates desiccation, as well as high and low temperatures, for which reasons the various methods of transmission of infection play no unimportant role in the epidemiology of diphtheria.

Pathogenesis. The bacillus can settle in any tissue. Most frequently it affects the *mucous membranes* (of the fauces, pharynx, larynx, nose, genitals), the *skin* if its epithelium is damaged (diphtheria of wounds, ulcers, intertrigo) and the *conjunctiva*. Reproducing at the point of entrance the diphtheria secretes a *poisonous toxin* which is spread by the lymphatic and circulatory systems.

All the clinical manifestations of diphtheria, local as well as general, are the result of the action of this toxin.

At the *site of its formation* the toxin produces an inflammation, most frequently with a fibrinous exudate (film), edema of the surrounding parts involving the adjacent lymph nodes.

Entrance of the toxin into the blood stream causes various degrees of general intoxication. Carried through the organism the toxin is absorbed by the cells and particularly easily damages the heart in which it provokes a process of inflammation and degeneration; then it affects the nervous system, the vegetative-endocrine system (the adrenals, in particular), the liver and kidneys, which accounts for the rapid development of *cardiac weakness*, *nephrosis* and *paralyses* in diphtheria.

Of great importance during convalescence is the ability of the organism to accumulate antibodies—*antitoxins*—which neutralise the poisonous toxins secreted by the bacillus.

The presence of antitoxins in the blood also accounts for the fact that some people do not contract diphtheria even when in contact with diphtheria patients and when the bacillus penetrates to the mucous membranes.

The presence of such antibodies in the human blood can be demonstrated either by experiment on animals or by the *Schick skin test*.

Schick has demonstrated that in persons who recently recovered from diphtheria or are insusceptible to this disease an injection of very small amounts of the diphtheria toxin (1/40 of the dose lethal to a guinea pig weighing 250 g) into the skin, for example of the forearm, causes no inflammatory reaction because the toxin is immediately neutralised by the antibodies (antitoxins) existing in the organism. If the organism does not have such antitoxins and is susceptible to infection, the toxin is not bound by anything and exerts its typical action on the tissues, characterised by *inflammation, redness and swelling* at the point of administration of the solution (0.2 ml), in which case the *reaction* is interpreted as *positive*.

An enormous number of observations of the Schick test in different countries has shown that this test can serve as a method of singling out people susceptible to this infection. The effectiveness of inoculations is controlled by the same test, since after successful inoculation a *positive Schick reaction changes to negative*.

A negative reaction denotes the presence of at least 1/30 active units per 1 ml of blood.

Comparison of the curves of the Schick age reactions with age morbidity shows a total coincidence, i.e., in the ages most frequently affected by diphtheria a positive Schick reaction occurs in the greatest number of cases, while, for example, in nurslings, during the early months of their life, the Schick reaction is, in most cases, negative, which is due to the transfer of antibodies by the mother to the child through the placenta and her milk.

An attack of diphtheria confers temporary, yet lasting, immunity; cases of recurrent infection are rare.

Pathological anatomy. Changes at the site of entrance of the bacillus. Fibrinous inflammation (film, membrane) is typical. On the mucous membranes of the respiratory tract this inflammation is rather superficial (croupous), on the mucosa of the digestive tract (fauces, pharynx)—deeper (fibrinodiphtheritic), involving not only the upper layers of epithelium but also its deeper parts and connective tissue. The croupous membranes from the larynx, trachea and bronchi therefore easily disengage in the form of molds, whereas in the fauces they are firmly attached and dissolve in the process of retrogression (dissolution of the membranes). The tissues are edematous; in severe cases hemorrhages are not infrequent.

Cardiac changes. The heart is very greatly and frequently affected. The changes are manifested in myocardial degeneration (albuminous and fatty) and inflammation (hemorrhages, edema, proliferation of the cellular elements)—myocarditis.

These forms of myocarditis result in formation of connective tissue, in place of the destroyed fibres, and hypertrophy of the muscle. There is no endocarditis in diphtheria.

Changes in the nervous system. Diphtheria affects mainly the vegetative nervous system and the peripheral nerves; destruction of cellular elements and, in peripheral nerves, of the myelin sheaths is observed. The *adrenals* (the medullary substance producing adrenalin) are very easily and early damaged by the toxin, which accounts for the fact that patients sometimes die so soon as a result of vascular insufficiency and cardiac weakness during early stages of severe cases of diphtheria.

The *internal organs* (liver, kidneys) show signs of degeneration.

Clinical aspects. The most frequent forms are *faucial diphtheria* and *laryngeal diphtheria* (croup); in the order of frequency these are followed by *nasal diphtheria*.

The other most frequent forms of the disease are *ocular diphtheria*, *genital diphtheria*, *surgical diphtheria* and *umbilical diphtheria in the newborn*.

Faucial diphtheria. The incubation period is 2-5 days. The following forms are distinguished: *localised diphtheria* in which the false membranes do not spread beyond the tonsils; *diffuse diphtheria* in which the false membranes spread all over the fauces and not infrequently extend to the nasopharynx; *toxic or severe diphtheria* in which, in addition to extensive false membranes in the fauces, there are *edema of the cervical connective tissue* and general symptoms of severe intoxication (cardiac weakness, albumin in the urine).*

As to the severity of the disease, the localised form is considered mild, the diffuse—moderate, and the toxic—severe.

The localised form of the disease begins with symptoms of general moderate *jadedness*, headache, slight pyrexia and early (on the 1st or 2nd day) appearance of a whitish-grey false membrane *on one or both tonsils*. The hyperemia around the false membrane is not intense, which distinguishes diphtheria from *coccal anginas* and scarlet fever (Fig. 51).

The cervical glands swell moderately, and the general symptoms of intoxication are mild. The internal organs suffer no particular changes. Properly treated with serum the process in the fauces does not last long (2-3-5 days); the false membranes are resorbed, *complications are rare*, damage to the heart is slight and is manifested in irregular and slow pulse (from the 4th or 5th day of the disease), unclear first sound or slight systolic murmur at the apex and a drop in arterial pressure, i.e., symptoms of so-called infectious heart.

Complications in the form of *paralyses and damage to the cardiovascular system* (myocarditis) are likely to develop only in cases of late and insufficient administration of serum or in the absence of serum treatment. In such cases myocarditis and paralyses may result in death.

This group of localised forms includes the mildest forms of diphtheria in which the changes in the fauces are manifested only in *punctate* or *islet* membranes. These islets may be present on the tonsils and may be scattered all through the pharynx (*islet form*).

* Similar forms (toxic) occur not only in faucial diphtheria, but also in diphtheria of other localisation.

Lastly, faucial diphtheria may be characterised only by a *catarrhal inflammation* without any false membranes, but this is an extremely rare occurrence. Such cases are diagnosed on the basis of a bacteriological examination and simultaneous discovery of typical cases of diphtheria in the focus.

Diffuse form. The onset may be more violent and the general symptoms more severe. The false membranes soon extend not only to the tonsils, but also to the palatine arches, uvula, lateral and posterior walls of the pharynx, and the nasopharynx. The membranes are grey and denser than in localised forms of the disease. The cervical glands become enlarged to a greater extent, but there is *no edema of the cervical connective tissue*.

The process lasts longer (7-10 days), usually causes various degrees of cardiac disorders, most frequently between the 5th and 15th days (slowing down and irregularity of the pulse, drop in blood pressure, dilatation of the heart, weakening of the sounds, and enlargement of the liver); it may be accompanied by complications, especially in cases of late and insufficient administration of serum. Paralysis, nephrosis and myocarditis are the *toxic complications* observed; otitis, lymphadenitis, sometimes pneumonia and nephritis are the purulent complications. In cases of timely administration of the serum *mortality* is reduced almost to zero; if the serum is administered too late, death may be caused by myocarditis or paralysis and sometimes pneumonia.

Toxic or severe form. The onset may vary. The process begins either subacutely, simulating the preceding forms and then developing into the more severe form, or—and this happens much more frequently—symptoms of general intoxication emerge at once in the form of fever, vomiting, often recurrent, and weakness; *extensive false membranes* rapidly form and spread all over the fauces, pharynx and nasopharynx, often extending to the soft and hard palates; the faucial mucosa is dark-red and very edematous.

The edema of the fauces is so strongly pronounced that the uvula is compressed and jammed in by the tonsils, and sometimes folded dorsad; the posterior wall of the pharynx cannot be seen. Respiration is difficult (pharyngeal stenosis), stertorous; the voice is constrained and has a nasal inflection, the mouth is open. A peculiar sweetish, nauseating odour from the mouth is observed. In some cases there is a *sanious discharge* from the nose.

Edema of the cervical connective tissue on one side or both is the most typical symptom of the toxic form of diphtheria. The edema appears as a soft, pasty and painless swelling; there is no redness or inflammation of the skin, which distinguishes this edema

CLASSIFICATION OF DIPHThERIA

TYPE	SEVERITY			COURSE
	Mild forms	Moderately severe forms	Severe forms	
A. Isolated forms	Faucial diphtheria	Localised: a) catarrhal b) islet c) membranous (membranes only on the tonsils)	Diffuse (membranes on the tonsils and other parts of the fauces)	Subtoxic (transitory to severe) Toxic 1st degree 2nd degree 3rd degree Hypertoxic Hemorrhagic Gangrenous Laryngeal diphtheria + tracheal and bronchial diphtheria (descending croup); a) without symptoms of stenosis b) with symptoms of stenosis
	Laryngeal diphtheria	Without symptoms of stenosis	With symptoms of stenosis: a) unoperated cases, b) operated cases	A. Duration: 1. Acute 2. Protracted B. Character: 1. Uncomplicated 2. Complicated a) toxic complications (paralyses); b) pyoseptic complications; c) respiratory complications; d) cardiovascular complications: 1) functional (vegetative) disorders; 2) myocarditis; 3) disorders associated with the serum sickness C. Presence of the serum sickness a) early, late b) mild, moderately severe, severe
	Nasal diphtheria	Without symptoms of stenosis a) catarrhal-ulcerative b) membranous		
Ocular diphtheria			Affection of the palpebral conjunctiva	
Genital diphtheria	Affection of the vulva		Affection of the vulva and vagina	

Severity indices

	Other localisations	Surgical diphtheria, cutaneous diphtheria	General	Local
			1) Metabolic-vegetative syndrome (general and cardiovascular weakness) 2) Hemorrhagic syndrome 3) Nephrosis	1) Edema of the faucial mucosa and of the cervical connective tissue 2) Extensive dirty membranes reaching beyond the fauces
B. Combined forms*	Faucial diphtheria + laryngeal, nasal, etc.			3) Specific odour from the mouth

* The system which is predominantly affected is indicated first.

from the scarlatinal adenophlegmon—a red, dense and painful swelling. Three degrees of the toxic form are distinguished according to the size of the edema: first degree—edema reaching to the second cervical fold (middle of the neck); second degree—edema reaching to the clavicle, and third degree—edema extending below the clavicle and spreading over the chest. The picture of general intoxication is manifested in pallor, apathy, extreme debility and early appearance of nephrosis.

Affections of the cardiovascular system constitute the most dangerous complications of toxic diphtheria.

In the severest cases (hypertoxic form) circulatory disturbances are observed from the very onset of the disease and are associated mainly with affection of the central nervous system and particularly the sympathicoadrenal and neurovascular apparatus. The clinical picture is characterised primarily by symptoms of sympathicoparesis or paralysis, and especially of vascular insufficiency which is closely connected with a secondary emergence of cardiac insufficiency, while the anatomical affection of the heart muscle plays no decisive role at this period. It develops later, for the most part at the end of the first and the beginning of the second week, and is manifested in degenerative and inflammatory changes in the myocardium.

The clinical picture of developing myocarditis is as follows:

Usually, from the 4th or 5th day or somewhat later, the pulse becomes irregular, quick, small and of low tension, the blood pressure drops (to 70-60 mm and lower), the heart is dilated, the heart sounds weaken, the liver considerably enlarges, the child's face grows pale, the mucous membranes become cyanotic and the limbs cold. In the severest cases these symptoms rapidly progress, vomiting, abdominal pain, restlessness and anguish appear, and a gallop rhythm is auscultated in the heart. Sometimes a block develops with a sharp slowing down of the pulse due to impaired conduction.

In most cases the foregoing picture presages death which most frequently occurs between the 7th and 15th days.

In a number of cases, however, myocarditis does not attain such gravity and the patient's condition gradually changes for the better. The earlier myocarditis develops, the graver the prognosis.

Affection of the kidneys is very frequently observed in toxic diphtheria; nephrosis usually develops from the very first days of the disease and is the most characteristic; nephritis occurs much less frequently.

The clinical course of renal affection in diphtheria is not severe; it runs without clearly defined edema and without uremia. The symptoms terminate for the most part in the 3rd or 4th week, seldom later. Paralyzes constitute a very frequent complication of toxic diphtheria. Usually they are of a *peripheral type* and are based on toxic parenchymatous neuritis involving the grey matter of the spinal cord.

The paralyzes develop in a definite sequence in the 2nd, 3rd or 4th week of the disease. Paralysis of the soft palate occurs the most frequently and appears the earliest; during this complication the voice acquires a nasal twang and the liquid being drunk flows out through the nose.

Paralysis of the ciliary muscle during which accommodation disorders develop is a rather frequent occurrence; in these cases the child has difficulty fixing his eyes on objects and cannot read or distinguish small type.

The foregoing paralyzes are often combined with lesions of other nerves—the abducens, oculomotor and facial.

In cases of greater extension of paralyzes the latter involve the limbs, neck and back; the muscles of these parts grow weak, the child can barely move his arms and legs, hardly holds up his head and is unable to sit up or turn over on his side; the child's muscular co-ordinations are lost (ataxia) and his reflexes disappear.

Paralyzes of the respiratory muscles and the diaphragm are the most dangerous. In these cases pneumonia easily develops in connection with the respiratory disorders and for the most part serves as the immediate cause of death in paralyzes. In most cases the paralyzes run a favourable course and the patients completely recover. The duration of the paralyzes depends on their extent and severity and varies between two or three weeks and several months.

Central paralyzes developing in the 2nd or 3rd week in patients with toxic diphtheria occur much less frequently; they are caused by thrombosis of the cerebral arteries, most frequently the internal carotid artery or the artery of Fossae Sylvii.

Spastic paralyzes of the limbs and sometimes of half the body develop; in cases of their localisation in the left part of the brain *aphasia* (loss of speech) is the result. The outcomes are permanent paralyzes with contractures and atrophy, and not infrequently death.

Lethal results of faucial diphtheria are observed predominantly in the toxic, severe form. The results depend mainly on the time the diagnosis is made, the day of administration of the serum and the dose in which it is administered.

In the group of toxic faucial diphtheria we must distinguish as the gravest and luckily its very rare forms:

a) the *hypertoxic form* in which all phenomena develop with extraordinary rapidity and the disease may terminate in the patient's death in the first two or three days with symptoms of cardiovascular insufficiency;

b) the *hemorrhagic form* in which considerable hemorrhages into the skin and the mucous membranes are observed;

c) the *gangrenous form* in which the false membranes soon begin to disintegrate. This form occurs after other infections which preceded diphtheria (measles, whooping cough).

The research conducted in recent years has shown that not only the specific diphtheritic toxin but also the organism's increased sensitivity to it, due to preceding sensibilisation, plays a certain part in the genesis of the severest forms of the disease. All these forms are barely amenable to serum treatment and are responsible for high mortality.

A milder variety of toxic diphtheria is the so-called subtoxic form which is characterised by less extensive affections of the fauces, unilateral moderate edema of the neck and only its pastiness and lesser intoxication. The prognosis in this form of the disease in cases of timely treatment is, as a rule, favourable.

Laryngeal, tracheal and bronchial diphtheria (croup). Development of inflammatory changes in the mucous membranes of the respiratory tract, mainly of the *larynx* where the diphtheria bacillus may settle, gives rise to stenosis of the trachea, which produces respiratory difficulties.

Diphtheritic affection of the larynx and the underlying respiratory tract is usually called *croup* to distinguish it from the false croup observed in other infections.

Croup most frequently occurs in children from one to five years of age.

Clinical aspects. Croup may develop as a *primary* affection involving at once and exclusively the larynx and trachea, or accompany other localisations and occur simultaneously with the affection of the fauces and nose.

It is believed that the process most often begins in the nasopharynx where the affection may remain unidentified.

Three stages are distinguished in the picture of development of croup: first stage—*catarrhal*, otherwise known as the stage of croupous coughing (1-3 days); second stage—*stenotic*; third stage—*asphyxial*.

The disease sets in gradually, its onset sometimes simulating

ordinary influenza; it is characterised by moderate temperature rises and mild coughing. Subsequently the coughing grows coarser and dry, the voice assumes a hoarse and barking character ("crowing"), gruff and raucous and sometimes disappears altogether; and the patient develops *respiratory difficulties* (stenosis).

The mechanism of development of stenosis involves not only edema and fibrinous inflammation (false membrane) in the pharynx but also, and in larger measure, a reflex spasm of the pharyngeal muscles.

The most typical sign of the stenotic period is *stertorous respiration* as a result of the air passing through the narrowed glottis.

At first the patient finds it difficult to inhale and then also to exhale; the child's breathing becomes so loud that it is sometimes heard in the next room; the child grows restless and tosses in its bed, trying to find a position in which it may be easier to breathe. In the beginning the periods of respiratory difficulties are short, then they grow longer, the difficulties increase and the yielding points of the thorax (epigastrium, jugular fossa, supra- and subclavicular fossae) sink in. During deep inhalations the pulse weakens or disappears, the child becomes extremely restless, and cyanosis develops; if the child is not administered special aid, the process enters its third (last) stage—*asphyxial*—during which the respiratory difficulties result in carbon dioxide poisoning. Stenosis debilitates the child, the tone of the respiratory centre decreases, respiration becoming fast and shallow. The child grows sluggish, sleepy and ceases to toss in its bed; it turns pale and its pulse becomes small and barely palpable. Cold sweat breaks out on the forehead; now and then the child has attacks of acute asphyxia which may cause immediate death. In other cases convulsions may develop and the child dies after a rather prolonged agony with signs of exhaustion of the respiratory and circulatory centres.

The second and third stages last from several hours to 2-4 days. This course is usually typical of croup in which no surgical methods are used and the therapeutic serum is administered late and in insufficient quantities. In cases in which the serum is administered in due time and in adequate doses the process is often arrested at the outset and no stenosis develops.

In cases of progressive stenosis timely intervention—*intubation* or *tracheotomy*—may save the child from asphyxia and enable the serum to produce its effect.

In neglected cases the process may extend from the larynx to the trachea and bronchi and involve even their smallest subdivisions (diffuse or so-called descending croup). In these cases the

patient's condition rapidly grows worse, respiration becomes very fast and shallow, and symptoms of general toxicosis, dyspnea, pallor and cyanosis come to the fore, the picture simulating a severe case of pneumonia.

Complications and mortality. The main danger and cause of complications in *faucial diphtheria* is the action of the diphtheritic toxin on the organism, especially on the heart and nervous system, leading to the afore-described paralyses and myocarditis.

In *croup* the toxin does not affect the organism so intensely because the cylindrical epithelium in the respiratory tract is much more easily cast off in the form of membranes, the process shows no tendency to extend deep into the tissues, and the absorption of the toxin is weaker, for which reasons severe affections of the heart and paralyses are rarely observed.

The most frequent and dangerous complication of this disease, which usually also causes death, is *lobular pneumonia*. It is only in extremely rare instances that this complication can be caused by the direct action of the diphtheria bacilli; in the overwhelming majority of cases it is due to secondary infection (diplo- and streptococcal).

Some complications, for example, decubitus ulcers from the intubation and tracheotomy tubes, may develop in patients as a result of the operation—intubation and tracheotomy.

The decubitus ulcers may serve as the source of the development of sepsis.

The *outcome* of croup depends on the severity of the disease, the patient's age, the time of serum administration and timeliness of surgical intervention. The main cause of lethal results in croup is complication of this disease by pneumonia.

In the past mortality from pneumonia averaged 10-20 per cent. In young children this percentage is higher. Mortality among operated cases is higher because it is usually the gravest patients who are subjected to operations. Now that overall therapy of croup has been elaborated, including wider utilisation of physiological methods of influencing stenotic respiration and the use of antibiotics, mortality has perceptibly decreased.

Differential diagnosis. Diphtheritic croup may be confused with other processes.

False croup not infrequently develops in *influenza* and is a result of acute catarrh of the laryngeal mucosa. Unlike true diphtheritic croup, false croup develops much faster and the child often suddenly experiences respiratory difficulties (stenosis); this very frequently happens at night and greatly frightens the members of the family. The disease is marked by higher fever, catarrhs, conjuncti-

vitis, coryza and redness in the fauces. The voice is often unaffected. The process soon passes, usually in a few hours, without serum treatment, after a hot general or foot bath (39-40°C for 10 minutes), administration of bromides (a tea-, dessert- or tablespoonful of a 1 per cent sodium or potassium bromide three or four times a day) and alkali (2 per cent sodium bicarbonate in the same doses) with hot milk. Of some value for the diagnosis is the fact that in some children the attacks of false croup recur 2-3 times a year.

An acute laryngeal catarrh which may cause stenosis may also develop during the prodromal stage of *measles*. The process is characterised by other symptoms of the prodromal stage manifested in rather high temperature, coryza and conjunctivitis later followed by Filatov's maculas and a rash on the face. The treatment is the same.

Sometimes the disease runs a rather severe course owing to a deeper necrotic inflammation of the larynx (see *Measles*).

Such a necrotic process may at times involve the larynx also in *scarlet fever* and produce, although seldom, a picture of a stenotic process.

Such forms connected with eruption of vesicles and papules on the laryngeal mucosa are similarly rare in *chickenpox* and *smallpox*. Foreign bodies gaining entrance into the respiratory tract (sunflower seeds, pieces of apple peel, etc.) very often simulate the diphtheritic croupous process. It is very important to obtain an anamnesis which helps to ascertain the fact of a foreign body entering the tract, since this is usually attended with choking and even an attack of asphyxia. These attacks of asphyxia, especially in cases of a large foreign body, for example, a sunflower seed, may recur during the process because, being for some time mobile in the respiratory tract, the seed is every now and then jammed between the vocal cords.

In cases of foreign bodies in the larynx the voice is usually unaffected.

The treatment is surgical, i.e., a tracheotomy with a subsequent bronchoscopy and removal of the foreign body.

Lastly, the following diseases may also simulate the picture of croup:

1. *Retropharyngeal abscess*. This affection is characterised by retraction of the head, stertorous respiration, constrained voice and difficulties of swallowing; palpation with the finger reveals a swelling on the posterior or lateral wall of the pharynx.

2. *Laryngeal swellings* which differ from croup in that they are chronic. In rare cases *secondary syphilis* of the larynx with an eruption of papules on the laryngeal mucosa may produce a pic-

ture of croup and require surgical intervention. The treatment is specific antisyphilitic.

Other localisations and forms of diphtheria. Nasal diphtheria occurs in two forms. A sanious purulent discharge from the nose, often from one nostril, irritation of the skin about the nostrils and its erosion and ulceration are typical of the *ulcerative-catarhal* form. This form is most frequently observed in very young children and even in the newborn.

Older children exhibit *membranous forms* which often accompany other forms; examination of the nasal cavities with a speculum reveals in these forms a *fibrinous membrane* on the septum or the nasal conchae. Nasal diphtheria is a relatively mild form but dangerous in that it may be easily overlooked, whereas untreated it may take a prolonged course, cause the diphtheritic process to extend to the fauces, larynx and other organs, and serve as a source of spreading diphtheria in the community.

It should be remembered that a sanious discharge from the nose is observed in children with congenital syphilis, and in these cases the addition of diphtheritic infection must therefore be excluded.

Diphtheria of girls' external genitals is almost exclusively a secondary infection following diphtheria of other localisation; in young children it more frequently develops as a primary disease through infection by attendants—bacilli carriers—who perform their genital toilet.

Diphtheria of the external genitals is characterised by edema, swelling, discharge from the genitals and appearance of ulcerations covered with a dirtyish film on the vulva and vagina and enlargement of the inguinal glands; painful micturition, during which infants scream, is often observed.

Sometimes toxic forms of genital diphtheria occur with extensive membranes and edema of the labia and subcutaneous tissue of the groins, thighs and mons veneris.

Surgical or wound diphtheria is marked by the appearance of dirtyish membranes on the affected part deprived of epithelium. The tissues are edematous and the glands become noticeably enlarged. One of the affections of this type which not infrequently occurs is *umbilical diphtheria of the newborn*.

Lastly, a diphtheritic membrane may appear on any part of the skin devoid of epidermis (skin folds behind the ears, in the groins, and in places of intertrigo—so-called intertriginous form).

Besides membranes, cutaneous diphtheria occurs in the form of sluggish ulcers which rapidly disappear after treatment with antidiphtheric serum.

Ocular diphtheria is observed relatively rarely; it develops most frequently as a secondary affection in the presence of diphtheria of other localisation, but may also occur as a primary disease.

Two forms—*superficial* (croupous) and *deep* (diphtheritic)—are distinguished according to the intensity and depth of the process.

The latter form is dangerous in that the process involves the eyeball and may extend to the cornea, as a result of which vision is in some measure impaired. In this form, especially in cases in which there is additional purulent infection, panophthalmitis sometimes develops and the eye is subsequently lost.

In differential diagnosis of ocular diphtheria a possibility of membranous conjunctivitis of diplococcal and other etiology should be borne in mind.

Diphtheroids which morphologically resemble diphtheria bacilli may be mistaken for Löffler's bacilli.

Complications in all these forms are rare and are not so strongly pronounced, but they run according to the same type—*action of the toxin* on the heart and the nervous system (cardiac weakness, paralyses)—or the type of purulent complications.

Treatment and care. Important as timely specific serotherapy (see below), which is the basic method of treating diphtheria, may be good care and close watching of the patient; an adequate diet and an appropriate regimen also play a very important part.

The air in the patient's room must be *fresh*; the room must be very thoroughly aired several times a day, especially in winter. During the warm periods of the year the windows may be kept open all the time. The patient may be kept warm by application of hot-water bottles to his feet and by rational clothing.

Clean linens and a well-arranged bed are an important feature in the care of the patient. Considerable attention must be devoted to caring for the *mucous membranes*. The patient must *gargle his throat* several times a day with a 2 per cent boric acid solution, 1 per cent hydrogen peroxide solution, weak potassium permanganate solution, 0.85 per cent sodium chloride solution or even plain boiled water if his age and condition allow it.

The child must not be given poisonous substances (for example, potassium chlorate) for gargling because children usually swallow some of the liquid.

Very young children must be given to drink more often; particular care must be taken of the mucous membranes of their lips and nasal cavities. In severe cases the lips and gums must be cleansed with a 0.85 per cent sodium chloride solution and then coated with vaseline or vegetable oil.

If there is a discharge from the nose and the mucosa is swollen, two drops of a 2 per cent protargol solution are instilled in each nostril several times a day. In cases of intense swelling an adrenalin solution is used.

Rp. Sol. Adrenalini hydrochlorici (epinephrine solution)

1:1,000 1.0

Aq. destill. 9.0

MDS. 2 drops into the nose

Once or twice a week it is well to give the patients, except in the gravest cases, a warm bath (37°C) or a rubdown with warm water and alcohol. In severe cases a rubber ring is placed under the small of the back; thorough care of the skin is necessary to prevent bedsores.

In *conjunctival diphtheria* a dressing is applied not to the *affected eye but to the healthy one* to safeguard it against infection. The affected eye is washed with a boric acid solution or a 0.85 per cent sodium chloride solution, and a 1-2 per cent xeroform ointment is applied to the conjunctiva.

The duration of the child's confinement to bed depends on the form of the disease and the *condition of the heart*; in usual forms—at least 2-3 weeks, in toxic and complicated cases—much longer, depending on the clinical data.

The patient must be ensured quiet surroundings.

Children affected with a severe form of diphtheria involving cardiac weakness *must not sit up; they must be fed and given to drink in a recumbent position* and must be safeguarded in every possible way against unnecessary emotions and manipulations. They must urinate and defecate into a bedpan. In cases of constipation they must be administered an enema.

In severe toxic forms of diphtheria it is necessary, in addition to the serum, to administer intravenously a hypertonic glucose solution (10-30 ml of a 25-40 per cent solution depending on age) and give the patients vitamins in the form of nicotinic acid (0.01-0.02 b.i.d.) and ascorbic acid (0.1-0.2 t.i.d.) for a period of 2-3 weeks; some authors recommend administration of strychnine from the first days of the disease.

Complete physical and mental rest, good care and extreme caution in carrying out all medical measures are particularly important in the treatment of myocarditis. Cardiovascular agents must be administered according to indications and with due regard for the pathogenesis of the particular disorders. In addition to glucose, administration of cordiamine (nickethamine) in doses estab-

lished for children (see p. 503) is preferable to that of camphor. Strychnine is also recommended.

Rp. Strychnini nitrici (Strychnine nitrate) 0.01
Aq. destill. 100.0
MDS. 1 tea- or dessertspoonful b.i.d. or t.i.d.
during meals

Strychnine is also administered in cases complicated by paralyses. In these cases patients must be given vitamin B₁ (0.005 b.i.d. or t.i.d.). Since the conduction function of the heart is often disturbed in diphtheritic myocarditis, no digitalis preparations must be administered.

Serum treatment of diphtheria. The treatment of diphtheria is specific, i.e., it consists in administration of *antidiphtheric serum* (*diphtheria antitoxin*).

The method of serum treatment of diphtheria was proposed by E. Behring in 1890. The serum is prepared by gradual immunisation of horses with increasing doses of *diphtheria toxin*.

The blood of such horses is taken at certain intervals and the serum is separated, the *amount of antitoxin* per 1 ml being determined in it by testing it on guinea pigs. This amount is called an "antitoxic unit" and the number of units is indicated on the ampules dispensed for treatment.

An ampule usually contains 5,000-10,000 au. The serum is thus *dosed not in cubic centimetres but in antitoxic units and is a titrated antitoxic serum.*

Its action consists in neutralising the poisonous toxin secreted by the diphtheria bacilli, which, as was already stated, produces the aforementioned manifestations of diphtheria—false membranes, edema of the connective tissue, general intoxication, symptoms of laryngeal stenosis, myocarditis and paralyses.

The diphtheritic toxin acts with extraordinary speed and intensity, often inflicting irreparable damage to the cells of the organism; serum treatment is therefore aimed at *administering the antitoxin as soon as possible, in adequate doses and in such a manner as to ensure the fastest possible absorption of the serum.*

The serum must be administered to all patients as soon as the diagnosis is made.

The diagnosis of diphtheria must be made clinically; bacteriological examinations must serve only to confirm the clinical diagnosis. This is often forgotten and sometimes, despite the clear signs of severe diphtheria with edema of the cervical connective tissue, the serum is not injected because no growth of the diphtheria

bacillus has been found. Negative cultures in cases of clear clinical manifestations must not prevent administration of the serum, since this may sometimes cost the patient his life.

Nor must the serum be administered indiscriminately in every case of exudative angina (with false membranes) "just to be on the safe side", for the serum sickness (see the corresponding chapter) is not entirely harmless, especially if the serum is injected repeatedly; the diagnosis must therefore be made judiciously, taking into account all the data of the anamnesis, status and epidemiology.

However, if the toxic or diffuse forms of diphtheria or diphtheritic croup are suspected, the serum must be administered before the results of the laboratory analysis are known.

In some cases diphtheria bacilli can be discovered by *direct examination of a smear* from the tampon containing mucus; it is therefore desirable to make a rule of examining these "preliminary smears" (so-called bacterioscopic examination).

The bacteriologic examination, whose results are usually obtained from the laboratory within 12-24 hours (and sometimes within 48 hours), is of the greatest importance. It must therefore be recommended that the method of accelerated bacteriological diagnosis should be more widely employed; this method consists in the following: the material is secured with the aid of a specially prepared moist tampon which is inserted in a sterile test tube and placed in a thermostat for 4-6 hours, after which a smear taken from this tampon is examined under the microscope. A positive result may be obtained in 4-6 hours.

A tellurium test has gained currency in recent years as an approved auxiliary method of rapid diphtheria diagnosis. In this test performed directly on the patient the false membrane on the fauces or in the nose is swabbed with a tampon soaked in a colourless 2 per cent potassium tellurite solution; within 5-10 minutes the false membrane turns black or grey in all diphtheria cases. Failure of the membrane to turn dark within 15 minutes rules out diphtheria. A positive tellurium test is observed in 10-20 per cent of other forms of angina (Simanovsky's angina, mycotic angina).

Numerous observations show that the earliest possible institution of serotherapy of diphtheria, especially of its toxic forms, and of croup ensures effective treatment and serves to decrease mortality from diphtheria.

In particularly grave cases (for example, hypertoxic or hemorrhagic forms) timely treatment implies *not days but hours*.

The serum may be administered *subcutaneously*, *intramuscularly* and *intravenously*.

The simplest, widespread and very effective method by which the serum is very quickly absorbed and which should always be used is *intramuscular* administration, usually by injection of the serum into the extensors of the thigh.

When administered subcutaneously the serum is absorbed much more slowly, for which reason this method is hardly ever used.

Intravenous administration of the serum may produce *anaphylactic shock* (see chapter on serum sickness); when this method is employed, a serum devoid of preservatives is usually administered.

The *dosage* depends on the severity of the case. In milder cases (islet, localised form of faucial, nasal or surgical diphtheria) an initial dose of 5,000-10,000 u is administered.

In a number of cases this suffices, but it is often also necessary to administer the serum on subsequent days, at first in the same, and then in smaller, doses repeatedly (until the membranes and general symptoms have disappeared); the dose is about 10,000-15,000 u for the duration of the disease.

In moderately severe cases (diffuse diphtheria, croup without symptoms of stenosis, genital diphtheria, more diffuse surgical diphtheria, mild ocular diphtheria) the initial dose is 10,000-20,000 u. The injections are repeated daily, at first in the same and then in gradually decreasing doses, so that the total dose of the preparation administered in the course of the disease amounts to 25,000-30,000-50,000 u.

Severe cases (diphtheria with edema of the cervical connective tissue, grave cases of croup with symptoms of stenosis, severe ocular diphtheria, extensive affections in genital diphtheria) require *repeated* administration of *large doses twice a day* on the first days and then once a day until disappearance of the membranes, edema, symptoms of stenosis and general intoxication.

The initial dose is 15,000-30,000 u; in very severe cases it is administered twice a day. Then this dose is administered once a day and is subsequently reduced so that the total dose administered in the course of the disease (if injected daily) amounts to 50,000-150,000 u.

In advanced cases (after the third day) the dose of the serum is increased.

Development of the serum sickness is no hindrance to administration of the serum. Administration of the serum must be suspended only in cases of *very severe serum sickness*; at any rate

It is desirable that the total required dose of the serum should be administered during the first 4-6 days of the disease—before the onset of the serum sickness.

During the serum sickness, if it is mild, the serum may be administered, but preferably subcutaneously. Addition of 0.5-1 ml of adrenalin (in a 1:1,000 dilution) per dose is recommended.

If the anamnesis contains indications that the patient had at one time already been administered serum, it is necessary to employ *Besredka's method* of serum administration, since repeated injection of serum creates the danger of *anaphylactic reactions*; this method consists in the fact that one and a half to two hours before administration of the serum the patient is given an intramuscular injection of 1 ml of the serum, the remaining requisite amount being administered after the aforementioned period. *Besredka's method* is desirable in all cases of serotherapy.

This method of desensibilising the organism prevents *shock* and severe *immediate* reactions and considerably diminishes the gravity of manifestations of the serum sickness.

Administration of dialysed, more concentrated serum cleared of ballast proteins and containing more antitoxin per 1 ml is recommended.

A purified serum known as "diaferm" (dialysed and treated with a ferment) is now being produced in the U.S.S.R.; its administration has proved highly effective, the serum reactions decreasing more than 50 per cent and becoming mild and brief.

Treatment of diphtheritic croup. Croup patients must be placed in thoroughly and repeatedly aired wards (with windows and transoms which can be opened). They must have an atmosphere of rest and quiet and experienced personnel so that they may be very closely watched. To prevent stenosis which is largely connected with laryngeal spasm, in cases of restlessness the child is administered hypnotics and sedatives—bromides and bromural.

Hot general (38-39°C) and local (foot) baths and hot drinks afford some relief.

Croup patients are no longer placed in special steam rooms as was done before.

Cases of pneumonia, as well as those suspected of pneumonia, require sulfa drug and antibiotic therapy.

Proper care and timely serum treatment make it possible, in a number of cases, to prevent development of severe stenosis and thus often obviate the necessity of surgical intervention. In other cases the process develops just the same and the patient cannot be saved without *surgical intervention* which is aimed at prevent-

ing aggravation of the patient's condition and consists in introduction of special tubes into the larynx or trachea by means of *intubation and tracheotomy*.

Intubation. Intubation is indicated by the transition of the second stage of stenosis to the third stage in which the patient is unable to cope with developing asphyxia—the jugular and subclavicular fossae and the epigastrium become deeply retracted, the pulse disappears, cyanosis develops, a cold sweat breaks out, the child becomes extremely restless and shows signs of fear.

The operation consists in introduction of metal tubes into the larynx through the mouth by means of a special instrument—*intubator*, the tubes allowing of free respiration. The operation is bloodless; performed skillfully it offers the patient quick relief and saves his life.

The instrument consists of: a) an intubator by means of which the tube is introduced into the larynx, b) a set of tubes of different sizes, c) an extubator by means of which the tube is removed from the larynx during extubation, and d) a mouth retractor. The tube has a thickening (head) at the end, which holds it in place on the vocal cords in the larynx. The head has an orifice through which a silk thread is passed. While the tube is in the larynx, the thread is brought to the exterior through the mouth, is run over the ear and is fastened with adhesive plaster on the cheek. In extubation the tube is removed by means of this thread.

Each tube is numbered, the number corresponding to the child's age.

Intubation techniques. The child is tightly wrapped up (arms and all) in a sheet. One assistant sits down and puts the child on his lap, holding the child's legs with his knees and the child's arms with his hands and pressing the child to his chest so that the child facing the physician is unable to move. Another assistant stands behind the first and with both hands holds the child's head at the same time pressing against the cheek the mouth retractor introduced into the child's mouth. The head must be held upright without letting it tilt (Fig. 51).

The physician sits down, facing the child, introduces the left



Fig. 51. Holding child for intubation

index into the child's mouth and along the root of the tongue reaches the epiglottis, presses the latter to the root of the tongue and, feeling for the entrance to the larynx, introduces the tube with his right hand by means of the intubator into the larynx through the glottis. If the tube has been properly introduced, the characteristic sound of air passing through it is heard, the child begins to cough and not infrequently expectorates phlegm and scraps of membranes. By means of the intubator and the left index the tube is lowered into the larynx so that the thickened end rests on the vocal cords. Then the intubator is taken out of the mouth, the mouth retractor is removed, and the child is put to bed, the aforementioned thread being brought out to the exterior and fastened on the cheek. To prevent the child from pulling out the tube by the thread, the child's arms are splinted.

The tube is left in the larynx for two days and is then removed by the thread. This period usually suffices for the serum to take effect and dissolve the membranes in the larynx so that, when the tube is removed, the child's respiration is in a considerable number of cases already quite free. However, in some cases after a certain lapse of time the stenosis increases again and the *intubation must be repeated* (2-3-4 times); in such cases it is said that the patient "does not relinquish" the tube.

If it is impossible to achieve the final effect, i.e., enable the child to breathe without the tube, a second tracheotomy has to be resorted to, because repeated intubations and prolonged keeping of the tube in the larynx may produce complications, namely: a) decubitus ulcers on the laryngeal mucosa, which reintensify stenosis and create a vicious circle—the child cannot breathe without the tube and it is necessary to repeat the intubation which aggravates the decubitus ulcers, and b) formation of a so-called false duct, when the physician, failing to introduce the tube into the larynx and applying a certain effort in introducing it, breaks the mucosa and introduces the tube under the mucosa somewhere away from the central position. As a result the intubation proves ineffective and asphyxia develops, for which reason it is necessary immediately to produce a tracheotomy.

A *tracheotomy* is a bloody operation consisting in an incision of the trachea with a subsequent introduction of a tube corresponding to the child's age; this tube is also retained for several days until the membranes in the larynx begin to disappear under the influence of the serum.

Sometimes a tracheotomy must be made as a *primary* measure without an intubation; this is indicated in very *advanced* cases

with severe symptoms of growing asphyxia and, as was already stated, in cases of a false duct, repeated failure to introduce the intubation tube, appearance of vomiting at each intubation attempt, in toxic diphtheria with considerable faucial and nasopharyngeal edema when intubation does not facilitate respiration because the edema is above the tube; a *secondary* tracheotomy is made when the child "does not relinquish" the tube and the intubation has to be repeated again and again, and, lastly, when the child keeps hawking up the tube. Neither should intubation be attempted in cases of severe laryngeal decubitus ulcers or in descending croup. At any rate, when intubation is attempted a *tracheotomy set* and an oxygen tent should always be kept in readiness.

Care of the child after intubation. The child's respiration must be carefully watched. If the tube becomes obstructed by phlegm or a scrap of membrane, the nurse must remove the tube by means of the thread and immediately summon a physician. If the child *hawks up the tube* or breaks the thread with its teeth, it is necessary to summon a physician at once. In the latter case the physician removes the tube by means of an *extubator*. As was already stated, the child is given semiliquid food.

Care of the child after tracheotomy. It is very essential that the tube should be properly fastened on the neck with tape. The bibs under the tube and the gauze bandage covering the orifice of the tube must be changed and the interior of the tube must be cleansed, from time to time, from the phlegm with a clean sterile feather.

Prophylaxis of diphtheria. It is necessary to make an early diagnosis and isolate the patient. The room occupied by a patient must be disinfected. All cases of suspicious anginas, laryngitis and sanguinolent discharges from the nose must be checked bacteriologically. This is particularly important as regards children's collectives in which isolation, disinfection and quarantine must be carried out particularly thoroughly.

If diphtheria is discovered in a children's institution, cultures must be made from the fauces and nose of all children without exception, and the children must be given the Schick test. The bacilli carrying children, especially those with a positive Schick reaction, must be isolated and placed under special observation.

Children are discharged from the hospital only after disappearance of all clinical manifestations of the disease and double bacteriological check-up on diphtheria bacilli.

Despite the numerous agents and methods proposed for the control of diphtheria bacilli carrying (administration of gentian

violet, pyocyanase, erythrin, penicillin, peracetin, bacteriophage, quartz, X-rays, etc.) none of them can be considered sufficiently effective.

Absterion from the diphtheria bacilli after their prolonged carrying can be accelerated only by raising the general resistance of the organism (proper regimen, aeration, adequate diet with enough vitamins, especially vitamin C).

Specific immunisation. Although the method of passive immunisation by administration of the antidiphtheric serum to children, who were in contact with diphtheria patients and who, on examination, turned out to be bacilli carriers with a positive Schick reaction, is sometimes used, it is not sufficiently reliable and offers no advantages since it confers brief immunity (only for 1-3 weeks) and increases the child's sensitivity.

This method may be employed only in exceptional cases in which it is impossible to ensure proper medical observation of those who were in contact with diphtheria patients (communities far removed from medical centres).

The method of *active immunisation* by inoculation with anatoxin is much more reliable. Anatoxin is a nonpoisonous variety of toxin treated with formalin, owing to which it loses its toxic properties; administered subcutaneously it causes formation of antibodies.

Immunisation with anatoxin is conducted in the form of two vaccinations with a 20-30-day interval (anatoxin doses of 1-2 ml), a single primary revaccination within 3-6 months (dose of 1 ml) and three subsequent revaccinations (same dose of anatoxin) at 3-4, 7-8 and 11-12 years of age (for details see *Supplement*).

After the inoculations the Schick reaction usually changes from positive to negative.

The extensive material on active immunisation accumulated in the Soviet Union and abroad shows that *this is a very effective method of controlling diphtheria*. It is necessary to inoculate all children, the most susceptible contingents—between one and eight years of age—in the first place.

By order of the U.S.S.R. Ministry of Public Health children from 5-6 months to 12 years of age are subject to compulsory antidiphtheric inoculation, whereas children 13 years old and older are inoculated only in cases of epidemic indications in the given locality. Inoculation of sufficiently large numbers of children prevents development of epidemics of diphtheria.

The incidence of diphtheria among inoculated children is lower and the disease runs a milder course.

In the U.S.S.R. the antidiphtheric inoculations are being administered since 1957 simultaneously with a pertussis-diphtheria vaccine against whooping cough. This vaccine is a mixture of detoxicated Bordet-Gengou bacilli suspension and purified concentrated diphtheria anatoxin (40,000 million Bordet-Gengou bacilli and 60 u of diphtheria anatoxin per 1 ml). The vaccine can be preserved for 18 months. It is administered subcutaneously in the subscapular region in a dose of 0.5 ml three times at intervals of 3-4 weeks; revaccination is performed with a dose of 0.5 ml at the age of 14 months and three years.

The experience of administering this vaccine has shown that it ensures complete immunity to diphtheria and is sufficiently effective against whooping cough. The reactions observed in a number of cases after inoculation were brief (not exceeding 24 hours) and were tolerated by children without any aftereffects.

DYSENTERY

Etiology. Dysenteric diseases are caused by a group of inter-related bacilli. The *Bacillus dysenteriae* was for the first time isolated in a pure culture by Grigoryev (1891) and was later studied in detail by Shiga (Japanese) and Kruse (German). The existence of other species of dysentery bacilli was subsequently demonstrated, the following being the most important ones: Flexner's bacillus, Sonne's bacillus, Hiss' bacillus, Schmitz-Stutzer's bacillus and Newcastle's bacillus.

The Grigoryev-Shiga bacilli break up glucose and levulose and secrete a very poisonous exotoxin. The other species of dysentery bacilli do not possess these properties and their toxic action is connected with the presence of endotoxins in them. Clinical, microbiological and epidemiological studies of various dysenteric diseases and outbreaks of dysentery have shown, however, that the toxicity of the microbe in culture not always directly corresponds to its effect on the patient's organism. A number of factors, the state of the organism and the conditions of its life in the first place, influence the character and degree of the manifestations of dysenteric infection to a much greater extent than do the properties of the microbes.

Epidemiology. Dysenteric diseases occur in all countries and at all times. The outbreaks and spread of dysentery are in very large measure due to poor sanitation and hygiene. Dysentery epidemics were observed in wartime, in connection with famine and natural

calamities, in congested areas with masses of people lacking a water supply and sewerage system, good drinking water and adequate food. Dysentery takes a considerable toll among children, particularly up to three years of age. The epidemics increase in summer and autumn (July-September) because children become overheated, foodstuffs, especially milk, spoil more easily in summer and the infection is spread by flies.

The feces of dysentery patients and the carriers of dysentery bacilli constitute the source of infection. Contamination of the hands, foodstuffs and the things used in the care of patients leads to the spread of the disease. Very dangerous epidemiologically are patients with a mild, inconspicuous form of dysentery which frequently remains undiagnosed, and patients with a protracted course of the disease who, while exhibiting an atypical picture, are a reservoir of the bacilli and may cause an outbreak of an epidemic.

Pathogenesis and pathological anatomy. The most important part in the pathogenesis of dysentery and the course of the disease is played by nervous disorders. This is confirmed by clinical observations, as well as anatomical and experimental data. The dysentery toxin is a neurovascular poison. It acts on the nervous receptors and its action is transmitted to the higher parts of the central nervous system, the changes in the regulatory function of the nervous system conditioning the multiformity of functional pathology in dysentery. The dysentery bacillus enters the intestinal tract through the mouth, multiplies and secretes a poisonous toxin which is absorbed into the blood and causes general intoxication—pyrexia, cardiac weakness, nervous symptoms manifested in headaches, sometimes clouded consciousness and convulsions. Excreted through the lymphatic vessels of the *large intestine* the toxin produces an inflammation in it, most frequently of a fibrinous character—either more *superficial (croupous)* in mild cases, or *fibrinous-necrotic*, diphtheritic in severe cases. It is these inflammations that cause the well-known grave intestinal symptoms. The inflammation may be catarrhal only in very mild cases and is most frequently observed in very young children.

The internal organs (heart, liver, kidneys) show degeneration caused by toxicosis. An attack of the disease confers temporary immunity only to one species of dysentery bacillus and no others.

Clinical findings. The incubation period is 1-7 days. The onset of the disease may vary. In some cases it is violent, with high fever, headache, vomiting and sometimes convulsions. Intestinal

symptoms in the form of a liquid stool with mucus and blood appear only several hours (sometimes 24 and more) later.

In other cases the onset is not acute, i.e., the general symptoms are feebly marked, the temperature rises but slightly and the process manifests itself in continuously increasing intestinal disturbances—pains and a frequent mucous and bloody stool.

At the *height of the process* the disease exhibits a number of characteristic symptoms—general *intoxication* and typical intestinal phenomena.

The *temperature varies* but, as a general rule, it is rarely very high. Sometimes the temperature is low even in the severest forms of the disease. The patients are generally jaded, somnolent and have headaches; young children are restless; in severe cases convulsions and unconsciousness are observed. The pulse is quickened; the graver the process, the quicker the pulse and the longer the acceleration persists. Various degrees of hyperleukocytosis with a neutrophil shift are observed in the blood. *Dehydration and loss of turgor* by the tissues occur very soon, especially in severe cases; the face is pinched, the eyes are sunken and the skin fold cannot flatten out. The mucous membranes are dry and the tongue is coated.

A characteristic *change in the stool* is the absence of fecal masses and presence of *mucus and blood*. The more severe the case, the sooner blood appears and the feces disappear and are replaced by mucus.

In the severest cases the stool resembles coffee grounds or liquid kitchen refuse with small scraps of mucus.

Severe twinges of pain (tenesmi) stimulating an urge to defecate and accompanying defecation appear; grave cases are attended with constant gnawing abdominal pain.

Palpation shows the abdomen to be retracted and painful. The intestines are palpated as painful cords (spasm). This symptom most frequently manifested in the sigmoid flexure of the colon is observed mainly in older children and very rarely in younger children.

Not infrequently the anus soon begins to gape and through its open sphincter the edematous inflamed rectal mucosa can be observed; this may subsequently be followed by rectal prolapse.

Course and forms of the disease. Diagnosis. Dysentery may occur in forms varying very widely in their course and severity—from the mildest to extraordinarily severe, showing extreme in-

toxication and ending lethally in one or two days (hypertoxic form), or involving an extensive necrotic process in the intestines; the disease may run a brief abortive course or a protracted course.

Diagnosis of atypical cases of dysentery, especially of so-called inconspicuous and dyspeptic forms, in young children may present considerable difficulties.

A bacteriological diagnosis is made by planting the dysentery bacilli from the patient's feces in nutrient media, differentiating them in sugar media and agglutinating them by corresponding agglutinating serums.

Positive cultures produced under ordinary hospital conditions are found in 20-30 per cent of the cases, but with proper techniques of obtaining the material and thorough laboratory examination the frequency of positive results reaches 60-70 per cent.

It should be emphasised, however, that the *diagnosis must be based mainly on clinical and epidemiological data*. Hence, any *acute colitis*, especially if it develops in the summer, with symptoms (even if not strongly pronounced) of intoxication, with mucus and blood, or even only with mucus, must be considered a *form of dysentery* regardless of the *negative results of the cultures*. The agglutination reaction produces a high percentage of positive data even in the early stages of the process.

Of course, even the mild cases in which the *dysentery bacillus has been found* must also be regarded as dysentery. In the presence of mild cases of the disease occurring as a focus (in separate collectives, among the population of one house, etc.) the diagnosis of dysentery is also very plausible. This is particularly important because it makes it possible to carry out prophylactic and antiepidemic measures, especially in collectives.

Of the other laboratory methods which are of no little importance in diagnosing dysentery, coprological examinations (microscopic examinations of the feces) and the agglutination reaction (so-called dysentery Widal test) are widely used.

In some cases of prolonged diarrhea with mucus in the feces microscopic examination reveals amebas (amebic dysentery) or *Giardia lamblia* (giardiasis). Microscopic examination of the feces for these Protozoa plays an important part in differential diagnosis of bacillary dysentery.

It must also be taken into account that acute gastrointestinal diseases in children, especially at an early age, may be caused not only by the dysentery bacillus but also by other pathogens, the importance of some pathogenic serotypes of the colon bacillus and *Salmonelleae* having lately been determined. Differential diagnosis of such diseases is often difficult and therefore requires bacteriological and serological examinations which make it possible to reveal their etiology.

In moderately severe cases all the symptoms develop within 3-4 days and the disease lasts 2-3-4 weeks.

The manifestations of initial intoxication begin to disappear (on the 4th or 5th day and sometimes sooner). As a result of intoxication, however, symptoms, very characteristic of dysentery, remain; these are *bradycardia* (slowness of the heart) which devel-

ops on the 5th or 6th day (in severe cases—later) and a certain, quite distinct drop in blood pressure. By the end of the 2nd or the beginning of the 3rd week the heart resumes its normal functioning.

In the beginning of the disease mucus and blood appear in the feces, then the feces disappear and reappear in the beginning of convalescence. The graver the case, the longer the period in which there are no feces at all. The number of defecations in moderately grave cases is 15-30 per day. Subsequently this number diminishes, the defecations become regular and the feces contain no pathological admixtures. Simultaneously with these manifestations the pains and tenesmi decrease, and convalescence begins. Poor nutrition and not infrequently a diminished *turgor of the skin* persist for a long time, especially in young children.

In *mild cases* the intoxication is very slight, the stool is not very frequent (5-10-15 times a day), the feces do not disappear altogether, there is not very much blood, no independent pains, the tenesmi are not intense and the cardiovascular disturbances are negligible. Often there are no complications at all, the nutrition is scarcely disturbed and the turgor is barely affected. The disease lasts 5-8-10 days.

Severe cases of dysentery produce various pictures of the disease.

In some cases (first group—severe form A, according to classification) marked symptoms of general intoxication with relatively moderate intestinal manifestations predominate. In these cases grave *nervous* symptoms come to the fore. The disease begins acutely with high fever, clouded consciousness, delirium and convulsions. Cardiac weakness and vascular insufficiency develop rapidly, there is a drop in the pulse and blood pressure, cyanosis appears, the limbs grow cold, the face becomes pinched, the eyes sink, the tongue and lips become dry, and the general tissue turgor sharply diminishes. At the same time there is a frequent mucosanguineous stool and gaping anus. In the severest cases of this group (hypertoxic) death with symptoms of collapse is possible during the very first days of the disease.

In other cases medical treatment gradually effects a detoxication and the disease takes a more favourable course which, however, does not exclude the possibility of complications.

In the second group (severe form B), in addition to general intoxication which does not develop to the extent described above, grave “local” (intestinal) manifestations come to the fore; extraordinarily frequent (“uncountable”) stool resembling coffee

grounds, excruciating abdominal pains, anal gaping and sometimes early rectal prolapse. In these cases the temperature most frequently sharply fluctuates and resembles the temperature obtaining in typhus and paratyphoid.

To exclude these diseases requires a repeated thorough bacteriological examination of the feces and later an agglutination test (Widal test).

This form may more frequently and sooner give rise to complications: pneumonia, otitis, antritis, mastoiditis, rectal prolapse, decubitus ulcers, desiccation and ulceration of the cornea, ulcerative and gangrenous stomatitis, and often general symptoms of sepsis. In these cases the prognosis is always serious and lethal outcomes are not infrequent, usually during later stages of the disease (10th-15th days and later). However, modern methods of treatment bring recovery in a number of even these severe cases.

The last, third group (severe form C) is, according to the picture of clinical manifestations, a mixed form; it exhibits the symptoms of the general above-described groups and must be regarded as the severest form of dysentery.

It should be noted that the development of complications in severe forms of dysentery is not only a result of general intoxication and grave intestinal affections, but is also connected with the development of a special state of altered reactivity (allergy).

The allergic state also not infrequently develops in milder forms of dysentery, especially in young children, and is marked by the appearance (on the 8th-10th-15th day) of secondary toxicosis, namely, worsening of the general condition, often fever, tachycardia, vomiting, and frequent stool. These easily add themselves to the aforementioned complications. Sometimes, instead of its usual duration (3-4 weeks), dysentery runs a subacute (up to six weeks) or protracted course in which the process operates for a longer time, now subsiding and now becoming aggravated, i.e., after brief intervals with an almost normal stool mucous feces reappear and not infrequently contain the dysentery bacillus. This course of the disease is most often observed in young debilitated children with dystrophy, rickets and exudative diathesis. A protracted course is also favoured by intercurrent infections (especially influenza and measles) and complications. Such cases are largely a result of untimely and inappropriate treatment. A prolonged course of the disease, sometimes lasting for months, justifies regarding these cases as chronic dysentery. Such patients constitute an epidemiological danger and are therefore subject to isolation;

they are placed for treatment in special institutions—sanatorium-type nurseries for chronic dysentery patients.

Complications. Some complications are connected with the action of the toxin. These are cardiac disorders (infective heart), metabolic disturbances with development of various degrees of dystrophy and hypo- and avitaminosis, with exalbuminous edema, and much less frequently—peripheral-type paralyses, serous inflammations of the joints (synovitis) and nephroso-nephritis.

Classification of Dysentery

Type	Severity	Course
Typical forms	1. Mild forms	A
A. With predominance of toxic phenomena	a) transition to moderately severe forms (A, B, C)	1. Acute (2-3 weeks)
B. With predominance of the local process	2. Moderately severe forms	2. Subacute (up to 6 weeks)
C. Mixed forms	a) transition to severe forms (A, B, C)	
	3. Severe forms (A, B, C)	3. Protracted (more than 6 weeks)
Atypical forms	Severity indices	4. Abortive (turning point of the disease between the 1st and 4th days)
	General Local	B
D. Forms with aggravated symptoms (hypertoxic form)	1. Meningoencephalitic syndrome	5. Without allergic waves and complications
E. Dyspeptic form (in nurslings)	2. Vegetative-metabolic syndrome:	6. With allergic waves and complications:
F. Inconspicuous forms	a) rapidly increasing general and cardiac weakness;	a) allergic type;
	b) early diminution of skin and subcutaneous tissue turgor, disturbance of water metabolism	b) septicopyemic type
	1. Character of the stool (absence of feces, branlike, brownish mucus	
	2. Permanent abdominal pain	
	3. Anal gaping and early rectal prolapse	

Other complications are connected with secondary microbial infection: otitis, antritis, mastoiditis, pneumonia, furunculosis and stomatitis (so-called septic ring).

Peritonitis and serous affections of the joints (synovitis) are rare complications. Young children often have pyelitis. *Rectal prolapse* is a frequent complication. In its milder forms it is manifested only in a slight protrusion of the mucosa on straining; in severe cases—rectal prolapse (*the entire rectum falling out several centimetres*).

So-called *exalbuminous edema* is another frequent complication. It is observed for the most part during the cold time of the year in emaciated children who were improperly fed (qualitative and quantitative starvation, avitaminosis). At first the *tissues grow pasty* and, in severer cases, extensive general edema and *cavitary hydrops* develop. They are caused by the action of the bacterial toxins from the intestinal tract and the products of abnormal metabolism on the epithelium of the vessels and the colloids of the cells. In these cases the kidneys are not affected and there is no albumin in the urine. The complication is very stubborn. As a complication of dysentery, exalbuminous edema now occurs much less frequently.

Very dangerous, too, is *gangrenous stomatitis* (noma) which leads to grave deformations of the face and often to death. Severe symptoms of dystrophy are observed in nurslings.

One more circumstance must be borne in mind. Sometimes, as was already stated, a case, which is not serious at first, shows considerable improvement towards the 6th or 7th day and then, suddenly, as though without any reason, changes for the worse—general intoxication increases, the stool becomes more frequent, complications (otitis, antritis, pyelitis, pneumonia) appear, the child grows extremely emaciated and may die. These aggravations ("waves") may take place earlier, on the 5th or 6th day of the disease, especially in young children. In these cases serum is ineffective. The cause of these aggravations is connected with the allergic processes in the organism (altered reactivity), owing to which new "waves" of the process develop, as is the case in other infections (scarlet fever, measles).

The *prognosis* depends on the patient's age and the form of the disease; it is the more serious, the younger the child, the more it has suffered from previous diseases and unfavourable developmental conditions, poor nursing and care, hypotrophy and rickets.

Treatment. Proper care and a hygienic regimen are the main part of the treatment. The patient must be given a maximum of *fresh air*, for which purpose the room must be continuously aired, while in summer the child must be taken outdoors. The airing of the room, however, must not expose the patient to cooling. Dysentery patients, especially severe cases, lose a great deal of heat, cool very easily and must therefore *be kept warm* by means of *hot-water bottles*.

It is very important systematically to give the patient *warm baths* (37-38°C); this also helps to preserve bodily heat, safeguards the skin against purulent coccal infection, to which the skin is very susceptible, and improves metabolism and perspiration.

A grave patient must lie on a rubber ring placed on a bedpan; he must not be allowed to sit up for defecation because this intensifies the straining and fosters rectal prolapse. In cases of pains and tenesmi the treatment consists in application of *heat to the abdomen, hot baths* (38-40°C), diathermy for the abdomen and ozocerite applications.

Care of the skin and mucous membranes is of enormous importance. It is important to *rinse the mouth* (2 per cent boric acid solution, weak potassium permanganate solution, boiled water); the mucous membranes (lips, tongue) must be coated with oil. Young children who cannot rinse the mouth should be given plenty to drink.

In severe cases with symptoms of dehydration, when desiccation of the bulbar conjunctiva easily begins, it is necessary to wash the eyes with a 0.85 per cent sodium chloride solution and *apply to the conjunctiva* a 2 per cent xeroform eye ointment, fish-liver oil or sterile vaseline oil.

Diet. The patient must be thoroughly and correctly nourished with due regard for his age, as well as the severity and stage of the disease. Starvation (both qualitative and quantitative) which is often the result of considerable dietary limitations and of a diet consisting mainly of carbohydrate food (cereal water, kissel) often does irreparable harm, causing emaciation, exalbuminous edema and noma. The diet must be adequate and must contain enough proteins, carbohydrates, fats, and plenty of vitamins; the foods must be easily assimilable. Only during the initial, acute stage, in cases of vomiting, a water diet is temporarily recommended (for 6-8 hours), following which the patient must be ensured an adequate supply of water and food.

During the acute stage the patient may be given milk with cereal water (2 parts of milk + 1 part of 4-5 per cent cereal water + 3-5 per cent sugar), concentrated cereal water (10 per cent), sour milk, tea with cream, farina (5-10 per cent), curds, kissel, and broth with egg yolk.

After the acute stage of intoxication the patient may be given the same food plus beef and vegetable soups with strained vegetables, cereals and quenelles, lean chopped beef, hard-boiled egg whites, zwieback, stewed fruit, puddings, and mashed potatoes.

The patient may be given this food despite the presence of blood and mucus in the stool.

The general principle of feeding children affected with dysentery is: be guided by the child's general condition and appetite and not by its stool.

Nursing is the basic method of feeding *infants*.

In very grave cases with marked toxicosis in young children (generally serious condition, repeated vomiting and meningoencephalitic symptoms) the diet must be prescribed much more cautiously (as it is done with respect to patients with toxic dyspepsia), namely, a starvation diet for 12-18, but not longer than 24 hours (depending on the child's condition) during which the child is given liquid, i.e., boiled water, tea, 5 per cent glucose solution, and Ringer's solution in a dose of 150 ml per 1 kg of his weight; later the child is given cooled human milk in small portions—10, 15 or 20 ml every two hours with an addition of a corresponding amount of liquid. Subsequently, the amount of food may be gradually increased, the child may be put to the breast for 3-5 minutes once or twice a day, the number of artificial feedings may be reduced to eight or even seven per day, and little by little nursing may be completely resumed.

If the child is on a mixed diet or is fed artificially and there is not enough human milk, the latter is replaced by mixtures, mainly sour (C-kefir, C-rice, buttermilk and diluted acidophilus milk), given at first in amounts one-half to two-thirds of those normally consumed at the particular age.

But these restrictive measures must not be carried out too long. As soon as it is possible, it is necessary to put the patient on a more adequate diet to ward off easily developing dystrophy.

During the period of convalescence the child is given cereals, kissel, soups, chopped beef and white bread; all through the illness, except the very first days, the patient must be given *vitamins* (in fruit juices and special preparations—200-300 mg of ascorbic acid, 5 mg of vitamin B₁, etc.) and small portions of fish-liver oil (nurslings—beginning with several drops and up to one teaspoonful, older children—beginning with one teaspoonful). In cases in which this method is used the process improves more rapidly, no emaciation develops and complications are less frequent.

In mild cases of dysentery children past one year of age may be given in the beginning of the disease grated fresh ripe apples—500-1,000 g per day in 5-6 feedings. This apple diet given to the child with plenty to drink for one or two days sometimes noticeably improves the stool.

Specific Treatment of Dysentery

Since 1939-40 chemotherapy has been the leading specific method of treating dysentery.

Sulfa drugs (norsulfazol [sulfathiazole], sulgine [sulfanylguanidine] and phthalazole [phthalylsulfathiazole]) were used for the most part until 1950-51; they were administered in doses of 0.2 g per 1 kg of the patient's weight per day, the daily dose being divided into six parts which were given at four-hour intervals. The course of treatment was 7-10 days; as soon as the general condition and local symptoms improved the dose was reduced to 0.1 g per 1 kg of the child's weight.

When sulfa drugs are used it is necessary simultaneously to give the child enough to drink; at the same time no ultra-high-frequency or quartz should be administered. The drugs may produce side effects (vomiting, cyanosis, restlessness, loss of appetite, polymorphous eruptions, leukopenia and neutropenia), especially if they are administered for a long time. The aforesaid phenomena necessitate immediate suspension of the treatment with the particular preparation.

During the first years of their administration in dysentery (1939-1941) the sulfa drugs proved therapeutically very effective. Subsequently, however, their effectiveness was observed to diminish, acquisition of resistance by the dysentery microbes to these drugs because of their very extensive administration in different diseases being considered one of the reasons for it. Sulfa drugs are the least effective in severe cases of dysentery, especially in very young children.

The antibiotics *synthomycin* (chloramphenicol) and *levomycetin* are used in the treatment of dysentery today.

Experience has shown these preparations to be very valuable since they produce a quick detoxicating effect and rapidly improve the intestinal symptoms.

Since their introduction (especially *synthomycin*) into extensive practice the use of sulfa drugs in dysentery has considerably diminished and they are now administered in mild cases to children past three years of age (in the absence of *synthomycin*). *Synthomycin* is administered per os four times a day in single doses of 0.02 g per 1 kg of the child's weight. The single dose for children weighing more than 15 kg is 0.3-0.5 g. In severe cases a shock dose (from one-half to the total daily dose in the course of 1 hour) is administered in the beginning of the disease; the course of treatment is 7-10 days. Since the preparations taste bitter, they should

be given to children in thick sugar syrup. The doses of levomycetin are half those of synthomycin. In cases of intractable vomiting synthomycin and levomycetin are administered in the form of suppositories (per rectum) in doses 50 per cent larger than those administered per os.

Biomycin (chlortetracycline) is also effective in the treatment of dysentery; this antibiotic is prescribed in a daily dose of 25,000 u per 1 kg of the patient's weight and is administered four times a day for a period of 7-10 days.

Administration of streptomycin can be recommended mainly for the treatment of infants in the first year of life in cases of secondary toxicosis.

This preparation is administered in a daily dose of 200,000-300,000 u dissolved in 20-30 ml of distilled water; it is given to patients in single doses of 1 teaspoonful 4-6 times per day for 7-10 days.

It should be remembered that administration of antibiotics may produce side effects in the form of eruptions and stomatitis. In cases of severe side effects (vomiting, loss of appetite, loss of weight, aggravation of intestinal symptoms) administration of the preparations must be suspended.

Treatment with antibiotics must be conducted under systematic control of blood and urine tests.

During the treatment with antibiotics the patient must be given enough vitamins, mainly of the B and C complex.

In addition to chemotherapy, stimulating therapy (blood transfusions, administration of plasma) is also very important in the treatment of dysentery, especially in very young children.

Intravenous administration of plasma in combination with glucose and ascorbic acid is very effective in controlling toxicosis (primary and secondary). In cases of marked toxicosis intravenous or subcutaneous infusion of plasma, glucose, Ringer's solution, vitamins B₁, C, etc., is indicated. Blood transfusion during toxicosis is contraindicated; it should be resorted to in cases of recurrent complications, protracted course of dysentery and developing dystrophy.

A favourable effect is produced by physiotherapy (diathermy in the abdominal region, ultraviolet irradiation, ozocerite; the latter acts as an analgesic and general sedative).

Formerly a serum was used in the treatment of severe dysentery during the first days of the disease. The serum does not produce an adequate therapeutic effect, especially in very young children. Serotherapy is now very rarely resorted to because of the effective treatment with antibiotics.

Symptomatic drug treatment is also of some importance in dysentery.

Cardiovascular disorders in severe forms of dysentery necessitate administration of cardiazol, cordiamine, caffeine and glucose in accordance with generally accepted indications. Since gastric and intestinal secretion diminishes, it is necessary to give dysentery patients gastric juice, pepsin, hydrochloric acid and pancreatin, especially in protracted cases.

Complications are treated according to general rules. In cases of otitis, antritis, mastoiditis and pneumonia the therapy includes administration of penicillin. In rectal prolapse the rectum is carefully repositioned and cold is applied to the anus; moreover, the child is not allowed to sit on the pot. In cases of synovitis—application of a splint and heat.

Gangrenous stomatitis requires irrigation with hot potassium permanganate, embrocation of iodoform, and administration of antigangrenous serum.

Exalbuminous edema indicates a diet containing enough full-value proteins (curds, meat, fish, eggs) and administration of vitamins A, B₁ and C in the form of fruit juices, ascorbic acid, fish-liver oil, yeasts and nicotinic acid.

Intramuscular injections of emetine (Emetini hydrochlorici 0.2, Sol. Ringeri sterilisati 10.0) in a dose of 1-2 ml twice a day for a period of five days are effective; the course is repeated four days after the end of the first course. Giardiasis is treated with acricine (quinacrine) in three cycles of seven, five and three days at seven-day intervals during which it is recommended to give the patient enemas of 1:2,000 rivanol (2-ethoxy-6, 9-diaminoacridine lactate) solution, as in malaria.

Since neurotrophic disorders play an important part in the genesis of dysentery with a protracted and so-called chronic course, in the treatment of this form of the disease it is necessary to concentrate most of the attention on increasing the general resistance of the patient, creating a favourable external environment and organising proper care and an adequate diet as factors which exert a normalising influence on the functions of the nervous system. A regimen with plenty of fresh air and appropriate health education are of essential importance in the total complex of therapy.

In addition to all the aforesaid, whenever indicated, it is necessary to administer stimulatory treatment (infusion of plasma, blood transfusion), physiotherapy and ferment therapy; special methods proposed for the treatment of protracted dysentery, Chernokhvostov's alcohol dysentery vaccine in particular, may be used in certain cases.

The method of administering the vaccine consists in injecting the preparation subcutaneously five or six times, beginning with a dose of 0.1 ml and gradually increasing it by 0.1-0.2 ml at 2-3-4-day intervals between the injections.

Intestinal disorders following an attack of dysentery with dysentery bacilli contained in the culture and prolonged excretion of bacilli without clinical manifestations are *indications for administration* of the vaccine.

The *contraindications* include age under six months, marked hypotrophy, aggravation of clinical manifestations and acute infections or contact with them.

In recent years (1952-1954) a combined method of immuno-chemotherapy (V. Troitsky) was recommended for the treatment of protracted and chronic dysentery and prolonged carrying of bacilli.

An enteral vaccine in the form of tablets (immunogen) is used as a preparation conferring immunity and a combination of two antibiotics (synthomycin and streptomycin) or as chemotherapeutic agents. The course of treatment is 23 days, administered in three cycles at 4-5-day intervals.

Prophylaxis. Early diagnosis and early isolation of the dysentery patient, as well as disinfection of the lodgings, linens and excrements, are important.

On discharging the patient it is necessary to repeat the bacteriological examination for dysentery bacilli.

In cases of disease in children's institutions every suspicious patient must be isolated and the premises disinfected (moist disinfection suffices).

Outbreaks of dysentery usually occur in summer and autumn, although cases are also observed in winter and spring, for which reason proper hygienic habits—cleanliness during meals, hand-washing, consumption only of boiled milk and water, rinsing of vegetables and fruit with boiling water before consumption—must be widely propagandised among the population. Destruction of flies is a very important part of preventing dysentery.

Considerable attention must be devoted to sanitation and hygiene in children's institutions. Good milk for children is one of the most important prerequisites of prophylaxis. Infant health centres, children's milk kitchens and children's institutions must be sure to provide only high-quality milk.

The prophylactic work persistently and consistently carried on by children's institutions and medical people in combating hypotrophy and rickets and in propagandising nursing is particularly important.

Specific prophylaxis. Active immunity is conferred by administration of Besredka's dry vaccine per os.

The child is given one-half or one pulverised dry vaccine tablet before a meal three times at one-week intervals; it is supposed that such vaccination can increase general and local tissue immunity. Enteral vaccination against dysentery is now considered ineffective and its extensive employment believed superfluous. The formerly widely practised phagoprophylaxis of dysentery is also ineffective. Bacteriophagy is recommended only in epidemic foci.

TYPHOID FEVER

Etiology. The causative agent of typhoid fever was discovered by Eberth in 1880. It is a short, thick bacillus, motile because of its flagella; it stains well with aniline dyes and is rendered colourless by the Gram method. It grows in usual media and, unlike the *Bacillus coli* does not curdle milk, does not form gas in sugar media, does not form indole or acid, and produces blue colonies in Konradi-Drigalsky's culture medium. The bacillus is easily agglutinated by the serum taken from a person who survived an attack of typhoid fever or from an immunised animal. It does not produce any exotoxin. Disintegration of the bacilli in the culture medium or in the human organism results in liberation of a very poisonous substance—endotoxin.

Epidemiology. Typhoid fever may be contracted in various ways. The patient himself is contagious because his feces, urine and saliva contain the typhoid fever bacillus not only at the time of the disease, but also long after recovery. Typhoid fever convalescents must therefore necessarily be kept under observation and checked for *bacilli carrying*. According to available information, 3.5 per cent of the people who survived an attack of typhoid fever long continue to eliminate typhoid fever bacilli (sometimes for many months and even years). These chronic carriers of bacilli are of considerable epidemiological importance.

The disease may also be contracted from apparently healthy persons who are *bacilli carriers*; while remaining healthy such a person may carry virulent bacilli in the intestinal tract and spread the infection.

Healthy people usually carry bacilli for a short time (several weeks) and are therefore of limited epidemiological importance.

Lastly, the microbes sometimes invade *the external environment* and infect the water (wells, river, water supply system), milk and foodstuffs, in connection with which mass outbreaks of the disease have been observed.

Such infection of the surroundings occurs particularly easily in cases of inadequate sanitation and hygiene, when the excrements of patients are thrown on the ground and no measures of personal prophylaxis are taken. This also occurs when external factors (wars, natural calamities) affect the sanitary state of an area.

An important part in spreading typhoid fever is played by flies who are in contact with the feces of patients and carriers of bacilli.

Typhoid fever epidemics are usually connected with failure to observe the rules of sanitation and hygiene; formerly they were very often observed in

wartime among troops and near the theatre of war among the population suffering from the aftermath of war, during famine, flood, etc.

Half a century ago typhoid fever was one of the most widespread infectious diseases in Europe. Planned regulation of the water supply and sewerage systems, food sanitation and systematic control of bacilli carriers during the last 40-50 years have sharply reduced the typhoid fever incidence.

Children contract typhoid fever through food, water and milk, if the milkmaid happens to be ill. Older children are affected more frequently, apparently because of wider contacts and more varied diet.

The highest typhoid fever incidence is observed at the end of summer, in autumn and winter.

Pathogenesis. Typhoid fever is a general hematogenic infection. It occurs through consumption of infected foodstuffs and water or by direct introduction of the bacilli into the mouth or intestinal tract by contaminated hands. Through the lymphatic system of the intestinal tract the bacilli enter the blood where they can be discovered by a culture of blood in bile during the first 7-10 days of the febrile period of the disease. Then, owing to the bactericidal properties of the blood, they disappear from the blood and settle in the lymphatic apparatus of the intestinal tract and gallbladder, intensively multiplying in the latter, since bile is their selective medium in which they can remain for a very long time and after recovery foster bacilli carrying.

The clinical picture is produced, on the one hand, by the action of microbial poisons (endotoxins) on the organism, since these toxins are absorbed by the blood and poison vitally important organs (nervous system, parenchymatous organs), and, on the other hand, by inflammatory changes in the intestinal tract, especially, its lymphatic apparatus. Owing to these affections, various microbes of secondary infection not infrequently gain entrance into the organism and cause purulent complications.

Pathological anatomy. Parenchymatous degeneration of internal organs (liver, kidneys and striated muscles; for example, Zenker's hyaline necrosis [also known as Zenker's hyaline degeneration] in which the rectus abdominis muscle undergoes coagulation necrosis) is characteristic of typhoid fever. Typical and peculiar are the changes in the lymphatic apparatus of the intestinal tract (follicles and Peyer's patches). The lymphoid tissue is replaced by an accumulation of large cells with a pale staining nucleus ("typhoid" cells) originating from the reticuloendothelium. These cells form "typhoid granulomas" which partly necrotise and produce ulcers which may bleed or become perforated. On recovery the ulcers cicatrise. The inflammatory process in the lymphatic apparatus of the intestinal tract thus goes through four stages:

cerebriform swelling of the follicles and, especially, the patches (their surface somewhat resembles the cerebral convolutions);

formation of ulcers—at first superficial and then gradually deeper ones which sometimes reach the serous membrane and may cause perforation of the intestinal wall;

cleansing of the ulcers and granulation;

cicatrization as the final stage of the typhoid process—the process in the intestinal tract.

In children these changes are usually much less pronounced than in adults, which in some measure also explains the more favourable prognosis in the cases of typhoid fever in children.

Clinical findings. The incubation period is about 2-3 weeks.

The disease usually sets in with symptoms of indisposition and *gradual* rise in temperature; in children, however, the process also develops in another manner, i.e., with a very high rise in temperature during the first two or three days, nausea and vomiting.

The *temperature curve* is quite characteristic and shows three periods: first—a gradually rising wave with 1.5-2° variations lasting about 4-5 days and sometimes a week; the second period also lasts about 5-7 days and produces a curve of a continuous intermittent type with variations of 1.5-2° (between 37.5° and 38.5° in the mornings, and 39.5° and 40°, sometimes higher, in the evenings). A gradual drop in temperature to normal begins at the end of the second week and lasts 5-7 days. In children the fever not infrequently ends with a series of extensive variations—between 37° and 39° and higher.

Thus the febrile period lasts a total of about 3-3.5 weeks. By the end of the first week, sometimes sooner, the patient shows a rather clear picture in which three symptoms are characteristic of typhoid fever—*intoxication*, a *rose-coloured eruption* and *enlargement of the spleen*.

The intoxication is manifested in headaches of which older children complain, sluggishness and sleepiness in the daytime, which in graver cases are followed by excitement and *delirium* at night, a state of profound *dejection* and somnolence at the height of the process, sometimes with total *unconsciousness*. In such severe cases not only general cerebral but also meningeal symptoms are often observed.

The eruption consists of rose-coloured spots (*roseola*) ranging in size from a pin-head to a lentil; the spots are not elevated or are but slightly elevated above the skin and disappear on pressure with a finger or stretching of the skin. The eruption appears at the end of the first or the beginning of the second week and lasts from several days to a week. It is a result of the general affection of the lymphatic system, which leads to development of small inflammatory foci around the typhoid fever bacilli in the lymph spaces.

The localisation of the spots is quite characteristic; they occur most frequently on the abdomen and lower part of the chest, then on the back, more seldom on the limbs and very rarely on the face and hairy part of the head. Usually there are very few spots (from 10 to 20), but sometimes the eruption abundantly covers the trunk and limbs, most of the elements appearing on the chest and abdomen. In some cases the eruption appears in successive stages. Sometimes there is no eruption at all.

The *spleen becomes enlarged* towards the end of the first week, sometimes a little earlier, and moderately dense. The enlargement is moderate—about 1.5-2 digits from under the ribs during inhalation—and persists about 1-2 weeks.

A coated, dry tongue covered with a crust at the height of the process is another characteristic symptom of typhoid fever.

The cleansing of the tongue always begins from the tip and the edges, for which reason a red triangle with the apex towards the root of the tongue is formed on its anterior half.

The *appetite* often diminishes or is completely lost, and the patient has to be forced to eat. In some cases, however, the appetite is retained all through the disease and increases during the period of convalescence.

The *patient's abdomen* is characteristic; at the height of the disease it is inflated and soft; palpation of the right iliac region produces a *splashing sound*. This symptom, like the coated tongue, distinguishes typhoid fever from tuberculous meningitis which sometimes exhibits a similar clinical picture.

In tuberculous meningitis the abdomen is usually retracted and the tongue is barely coated.

The stool varies. The diarrhea with liquid yellow feces resembling pea soup, typical of adults, is far from constant in children, although it is observed at the height of the process in a number of grave cases in which the patient has a stool 3-8 times a day. Children are most frequently *constipated* all through the disease, especially during the first week.

Very often the patient has bronchitis with a moderate cough which does not appear at once, but at the end of the first or the beginning of the second week.

The *cardiovascular apparatus* is moderately affected; at the height of typhoid fever the heart is slightly dilated, the sounds become dull and unclear; not infrequently there is a systolic murmur and a certain drop in blood pressure; however, severe disorders requiring prolonged administration of *cardiacs* are observed in children relatively rarely. Very characteristic is *bradycardia* which appears quite early and accompanies the entire febrile period.

The pulse slows down rather sharply and may be 60-70 beats per minute at a temperature of 39-39.5°. But this symptom is inconstant, especially in very young children, and is not infrequently observed only at the time the temperature begins to drop.

The blood shows leukopenia—6,000-3,000 leukocytes with a neutrophil shift to the left, disappearance of eosinophils and decrease in the thrombocyte count. The erythrocyte sedimentation rate at the height of the disease is high (30-50 mm per hour by Panchenkov's method).

Albumin and casts sometimes appear in the urine; the *diazoreaction* is positive from the onset of the disease to the period of convalescence.

Sense organs. Impaired hearing is quite characteristic at the height of the disease and during the first days after the end of the fever.

In virtue of general intoxication, diarrheas and nutritional difficulties the child often *loses a lot of weight*. A slight scaly desquamation somewhat resembling that which follows scarlet fever, but does not involve the palms of the hands and fingers, sometimes appears on the skin at the end of the process.

If no relapses or complications occur, the patient's condition improves in the third week. The temperature begins to drop, the consciousness clears up, an appetite appears and convalescence starts. During this period the hair is not infrequently observed to fall out, but subsequently grows very well again.

Relapses. The disease does not always run so uneventful a course. In some cases the temperature drops to normal in the third week, but suddenly, after one or two weeks of such a remission, it begins to rise without any apparent reason again, the state of intoxication recurs, the spleen enlarges again, an eruption sometimes appears, and the disease seems to return with all its symptoms. The temperature exhibits the usual curve again, sometimes with the same three-week course and sometimes a more rapid course. In some cases the first attack is mild, is of shorter duration and atypical, while during the relapse the disease takes a prolonged and severe course.

Relapses may also occur earlier without a break in temperature so that the second temperature curve does not begin after a break, but begins at the end of the first curve. The relapses are due to a secondary multiplication of the typhoid fever bacillus in the blood from which it disappeared but was retained in the gallbladder. Development of an allergic state of the organism with diminished immunity may play some role in the genesis of the relapses, as is the case in the fluctuating course of other acute infections.

Typhoid fever may vary in severity. All in all the disease is not severe in children, complications are not so frequent and mortality is not high (rarely more than 2-3 per cent).

However, in some cases severe forms of the disease may occur, especially in very young children; owing to complications, these cases most frequently end lethally.

Death is usually the result of complications—pneumonia, peritonitis and noma.

It should be noted that in nurslings and very young children the clinical picture of typhoid fever has a number of peculiarities and often lacks the characteristic symptoms. The diagnosis of these cases without bacteriological and serological examinations may therefore present considerable difficulty. Usually they have a more acute onset, the temperature reaches its maximum sooner, its curve is less regular, symptoms of enterocolitis often prevail, and in some cases apathy or restlessness and sometimes symptoms of meningism are observed.

Sometimes typhoid fever in children during the first year of life is mistaken for toxic dyspepsia, sepsis or dysentery and only after long clinical observation and numerous laboratory examinations is it possible to make a true diagnosis.

Complications. Respiratory tract—bronchitis which, in some cases, may persist for a long time, and focal-type pneumonia which usually sets in at the height of the disease. Pneumonia may give rise to *empyema*. Nosebleed is sometimes observed.

Digestive tract—stomatitis with noma as its gravest form.

This complication develops in very emaciated, debilitated children who have survived several diseases, especially if they had measles shortly before typhoid fever and were malnourished (lack of vitamins). These complications are now scarcely observed.

Infrequent but dangerous *intestinal hemorrhages* usually occur in older children. *Peritonitis* is also observed infrequently. Intestinal hemorrhage is characterised by an appearance of blood in the feces and their dark colour; rapidly developing weakness, pallor, and a drop in the pulse and temperature are often essential symptoms of intestinal hemorrhage. The symptoms of incipient *peritonitis* are—meteorism (gaseous distention of the abdomen), pains, very rapid pulse, vomiting and a sharp deterioration of the patient's condition.

Of the other complications suppurative parotitis, suppurative otitis, furunculosis, osteomyelitis, purulent arthritis and thrombophlebitis occur as manifestations of septicopyemia.

A rather grave picture in the nervous system may be produced by encephalitis and *meningoencephalitis*. They usually develop at the height of the process and are characterised by cortical and meningeal symptoms—unconsciousness, convulsions, paresis, aphasia, loss of hearing and vision and a meningeal symptom complex.

A lumbar puncture shows the cerebrospinal fluid to be either normal or to contain a small amount of albumin; the number of formed elements is increased, the cultures are sterile.

Despite the considerable gravity the *prognosis* in these cases is not always unfavourable; sometimes the neuropsychic functions may be completely restored; other cases may result in permanent paralyses, retardation of mental development, and cortical epilepsy.

A *bacteriological diagnosis* may be made in the first 7-10 days by means of planting blood in a bile medium in which the typhoid fever bacillus grows;

the bacillus is differentiated in Conradi's or Endo's culture medium or in sugar agar and is agglutinated by the serum of an immunised animal. A blood culture must be made on the very first suspicion of typhoid fever. During later periods the Widal test is performed. The serum is separated from the patient's blood, 1:10, 1:50, 1:100 and 1:200 dilutions of the serum are made and are mixed with the loop of a day's agar culture of typhoid fever, paratyphoid A and paratyphoid B. The agglutination reaction is characterised by a clumping of the bacilli. The test is performed in a small test tube or under the microscope.

The Widal test is considered demonstrative in 1:100 and 1:200 dilutions. Agglutination begins to appear in 10-15 days.

The results of this test are particularly convincing if its repeated performance reveals an increasing titer of agglutination.

It should be noted that in some cases the Widal reaction may be negative for a long time, especially in cases with a subsequent relapse. Moreover, the reaction may be positive in a patient who has survived an attack of typhoid fever or has been given a preventive antityphoid inoculation. It follows that the Widal test can play only an accessory diagnostic role.

Treatment, care, hygiene. Proper care is very important. The air in the patient's room must be kept fresh, for which purpose the room has to be thoroughly aired in any weather. The patient's bed must be placed so that the patient can be approached from all sides. If the child is in a serious condition, he must lie on a rubber ring. Older children must rinse their mouths with a 0.85 per cent sodium chloride solution, water, potassium permanganate or boric acid (2 per cent) solution. The lips and tongue must be coated with oil. Very young children must additionally be given a lot to drink.

Diet. Considerable attention must be devoted to feeding the patient. A starvation diet which was long considered compulsory for typhoid fever patients is now held to be *harmful*. The patient must be given *plenty of nourishing food*.

In the beginning, as well as at the height of the disease, the patient should be given liquid and semiliquid food. Children very well tolerate tea with milk or cream, sour milk, kefir, sour cream, curds and butter. They may be given cereals, jellies and fresh grated fruit and vegetables. The soup may be made of grated vegetables, cereals, butter, sour cream and an egg. Meat must be served in the form of quenelles in the soup or a small rissole. The patients may be given fish, zwieback. During the period of convalescence the diet may vary still more. Coarse food difficult to digest and assimilate must be avoided. It is very important to give the patients *vitamins*—fruit juices and fish-liver oil. Nurslings must be given *human milk*. The bowels must be kept open and enemas administered in cases of constipation. In cases of pain and meteorism applications of heat to the abdomen are recommended. Special attention must be devoted to the *skin* in view of the possible bedsores (it is well to place a rubber ring under the child).

The following general measures are beneficial: warm baths (37°C gradually reduced to 35°C), and in cases of fever—cold packs (34°) (one bath and one pack).

Drug therapy. Antibiotics (synthomycin, levomycetin, biomycin) are now effectively used in the treatment of typhoid fever. Caffeine is administered in cases of cardiac weakness. Typhoid fever patients must be given no camphor because they easily develop abscesses.

In treating children affected with typhoid fever attention must be concentrated mainly on observing the rules of hygiene, care and diet.

Sulfa drugs and penicillin are used in cases of pneumonia.

Complications are treated according to general rules.

Prophylaxis. The measures of social prophylaxis consist in controlling the sources of drinking water and the foodstuffs and in safeguarding them against contamination. It is necessary to watch convalescents and disinfect their feces; carriers of bacilli must be kept under bacteriological control. Early diagnosis of typhoid fever and hospitalisation of patients is of enormous importance.

Patients are discharged from hospitals after multiple bacteriological tests, the first test being performed one week after disappearance of the clinical symptoms, the subsequent bacteriological examinations being made at five-day intervals. If bacteriological control is impossible, the patient is discharged from the hospital two weeks after disappearance of the clinical symptoms (see *Supplement*). In cases of possible development of an epidemic specific vaccination with a typhoid fever vaccine is administered (heated or formalin typhoid or typhoid-paratyphoid A and B vaccines—monovaccines, divaccines and trivaccines—are used); three subcutaneous inoculations at 7-10-15, but not more than 20-day intervals are made or tablets of dry vaccine are administered per os in doses of 3-2-1 tablets (depending on the child's age) three times at one-week intervals.

PARATYPHOID DISEASES

Studies of the bacillus isolated from the blood, urine and feces of patients clinically diagnosed as typhoid fever cases have revealed that in some instances this bacterium differs from the typical typhoid fever bacillus as regards the agglutination reaction, although it belongs to the same general group, for which reason it has been named the *paratyphoid bacterium*. The most frequently occurring types are A and B. Clinically the course of such paratyphoid may in no way differ from typical typhoid fever caused by Eberth's bacillus, and as regards the pathogenesis and pathoanatomical changes paratyphoids A and B do not essentially differ from typhoid fever; it is therefore wrong, as it is not infrequently done, to regard atypical cases of typhoid fever as paratyphoid.

Paratyphoid can be diagnosed only bacteriologically, either by a culture of the bacillus from the blood, urine and feces, or by the Widal test. However, the paratyphoid bacillus may sometimes produce quite atypical forms of the disease running a short irregular course and sometimes even diseases clinically simulating enteritis and dysentery. Hence, the great importance of bacteriological examination of such atypical cases. Such paratyphoid diseases which simulate even sepsis most frequently occur in nurslings and very young children. Infection with the paratyphoid bacillus usually occurs through contaminated meat and other foodstuffs.

The treatment and prophylaxis are the same as in typhoid fever.

TYPHUS

Etiology. The establishment of the basic facts relating to the etiology and epidemiology of typhus is associated with the names of Russian scientists; thus, in 1876 Mochutkovsky (experimenting on himself) demonstrated the existence of the infectious principle in the blood of the typhus patient, while

G. Minkh established in 1878 that typhus was transmitted by lice. Today it is incontestable that the causative agent of typhus is the *Rickettsia prowazeki* named after the scientists Ricketts and Prowazek who discovered it in 1911-1913. The *Rickettsia* are minute organisms, $0.3 \times 0.4 \mu$, round or oval and most frequently arranged in pairs; they are found in the blood and organs of typhus patients during the febrile period (disappearing 2-3 days after the drop in temperature) and in the epithelium of the gastrointestinal tract of lice infected with the blood of typhus patients.

Rickettsia cannot be cultivated in artificial culture media; they multiply only in lice or the organs of experimental animals.

The *Bacillus proteus* X_{19} to which some authors recently ascribed an etiological role in typhus is a concomitant microbe, but so constant that it is agglutinated by the serum of typhus patients, for which reason agglutination of *B. proteus* X_{19} (the Weil-Felix test) assumes an almost specific character and is a very valuable method of laboratory typhus diagnosis.

The test is usually positive no sooner than the second week of the disease and only in rare cases on the fifth day of it.

Information has been obtained in recent years concerning the relationship between the *B. proteus* X_{19} and the *Rickettsia* (V. Krestovnikova). The existence of common receptors in the *Rickettsia prowazeki* and the *B. proteus* X_{19} is quite probable.

Epidemiology. Typhus occurs in the form of epidemics very largely due to unsanitary living conditions.

The disease is not directly transmitted from a patient to a healthy person. Typhus is contagious only because of the existence of an intermediate agent—the louse (the body louse as the most mobile).

The louse is not only the mechanical vector, but also the intermediate host since, after its infection with the blood of a typhus patient, the causative agent goes through the four- or five-day cycle of its development in the organism of the louse and only then (i.e., never sooner) is the louse able to transmit typhus to a healthy person during its bite and contamination of the wound with infected excretions; the infection can also be introduced by rubbing the excrements of the louse into the wound during scratching.

The knowledge of this incubation period when the louse is infected but is as yet unable to transmit the infection is of exceptional importance in carrying out epidemiological measures (hospitalisation and disinfection of the focus during the first days of the disease).

The virus in the organism of the typhus patient persists during the febrile period (and possibly during the last two days of incubation) and the organism is rid of it usually by the 3rd day of convalescence. However, epidemiological observations show that sometimes it may persist in the blood for as long as ten or twelve days after the drop in temperature; no *Rickettsia* are found in the excretions of the patients. The infected louse remains a carrier all through its life which, owing to the infection, is reduced to 30 days.

Since lice are the chief epidemiological factor in typhus, unsanitary living conditions which foster the spread of lice also favour development of typhus epidemics.

Extensive epidemics which affected the armies of belligerent countries and different countries after war and famine are well known. Outbreaks of typhus were often observed in tsarist Russia. The epidemics and outbreaks were caused by the very unsanitary living conditions of the needy, sections of the population characterised by lack of water, filth, insufficient clothing

and congestion, i.e., conditions under which *body lice* were easily able to multiply and be transmitted from person to person. Of course, the physical and psychic state of the people and their malnutrition also played an important part.

There were no typhus epidemics in the U.S.S.R. even during the hard years of the Patriotic War; since the end of the war typhus has occurred only as sporadic cases and isolated outbreaks.

Only older children are affected by the disease, i.e., those who have greater possibilities of contact and, hence, of infection; however, there have been cases of the disease affecting nurslings (after three months), usually when the mother contracted typhus.

The highest disease incidence is in late autumn and winter.

One attack of the disease confers rather stable immunity, although the disease is observed to recur, especially after long intervals of time.

Pathogenesis and pathological anatomy. The anatomical lesions consist in affections of the small arteries, veins and capillaries by a peculiar process with formation of thrombi inside the vessels, *lesions of the walls* and formation of *nodules around the vessels* (granulomas). The process subsequently ends in *obliteration of the vessels*. These changes occur in the nervous system, skin and muscles.

Affections of the heart (frequently—interstitial myocarditis, less frequently—degenerative processes) and of the endocrine glands (adrenals, hypophysis) are also observed.

The grave disorders of the cardiovascular system are caused, in addition to intoxication with microbial poisons, by affections of the adrenals and other endocrine glands.

Owing to the physiological factors of age, these symptoms in the cardiovascular system of children are less clearly pronounced, which possibly accounts for the milder course of typhus in children.

Clinical findings. The incubation period averages 11-14 days but may be from 7 to 20 days. The disease rarely sets in suddenly; usually headaches and increasing weakness develop for a period of 2-3 days. Chills and vomiting are rare. The temperature rises steeply, and on the 3rd or 4th day the disease usually reaches complete development and exhibits the three main symptoms—*general intoxication*, *eruption (roseola)* and *enlargement of the spleen*.

The intoxication is manifested very clearly—intense headaches, pains in the muscles and hyperesthesia. The last symptom is very characteristic of typhus—the child begins to moan and whine if you compress its thigh in several places along the course of the nerve bundle. Not infrequently the child becomes *delirious* and in severer cases the child's *consciousness is clouded*. *Depression* predominates in the picture of the disease, while excitement occurs seldom.

The temperature is quite peculiar; rising steeply to 39.5-40° by the 3rd day it persists with different degrees of variation to the 8th-10th day and then usually drops with a *brief lysis*. Abrupt, rapid rises in temperature and its critical drop are observed rather rarely. The febrile period lasts a total of 12-14 days.

The eruption usually appears soon—on the 3rd or 4th day, seldom on the 5th or 6th day; it breaks out rapidly and is for the most part abundant, which distinguishes typhus from typhoid fever in which the roseola does not appear before the 7th or 8th day, breaks out gradually, over a period of several days, is scant and does not spread.

A roseola consists of rose-coloured spots which are not elevated above the surface of the skin or are elevated but slightly, have unclear contours and are the size of a millet grain or small lentil. It is particularly frequently localised on the external surface of the forearm, chest and abdomen, more seldom on the face and very rarely (if the eruption is abundant) on the hairy part of the head. The number of spots varies. They become pigmented rather soon, assuming a brownish colour, and disappear in about a week.

In severe cases the roseola appears simultaneously with *petechiae*.

The *spleen* begins to be palpated on the 3rd or 4th day, which also distinguishes this process from typhoid fever in which the enlargement of the spleen is not manifested before the end of the first or the beginning of the second week. The spleen is of average size—usually it protrudes one and a half to two digits from under the ribs, unlike the much more sharply enlarged spleen in *relapsing fever*. It is of average density, and softer in severer cases.

The skin is dry, there is no perspiration, and a slight desquamation is sometimes observed at the end of the process. The glands are not enlarged. There are no catarrhs, and only in graver cases is a *sharp filling of the conjunctival vessels* observed. The tongue *becomes dry and heavily coated* rather early. The appetite diminishes but is rarely lost altogether, so that the child can usually be adequately nourished.

The stool is normal or a certain tendency to constipation is observed. The abdomen is usually soft and not inflated.

A slight cough appears in severer cases. The pulse is usually *accelerated* all through the febrile period and becomes somewhat slower and irregular only after the drop in temperature; this distinguishes typhus from typhoid fever with its bradycardia which is very characteristic of it at the height of the process. The heart is usually dilated at the height of the process, the heart sounds are dull, and a drop in blood pressure is observed. These symptoms are rarely very intense and, constituting no danger to life, seldom require administration of cardiacs to children, which sharply distinguishes typhus in children from that in adults, cardiac complications being quite frequent in the latter.

In severe cases albumin is sometimes found in the urine. A positive *diazo-reaction* of the urine is characteristic of these cases.

The *blood* shows leukocytosis of a neutrophilic character (10,000-20,000); the drop in leukocytosis somewhat precedes that in temperature: the erythrocyte sedimentation rate is not high (10-20 mm per hour).

In addition to the afore-described picture, the following signs are also of some importance in the diagnosis of typhus: 1) dyspnea without pulmonary symptoms, 2) injections (more rarely hemorrhages) into the conjunctiva, 3) depression and somnolence in children under three years of age; excitement and delirium from time to time in older children, 4) positive diazo-reaction, 5) quickened pulse, 6) first heart sound dull, 7) drop in temperature on the 4th-8th-12th day of the disease, and 8) low erythrocyte sedimentation rate (10-20 mm per hour).

The complications are few and rarely severe; bronchitis is the most frequent complication, pneumonia occurs more seldom, and stomatitis is sometimes observed; nurslings and very young children have dyspepsia and colitis. Some cases are complicated by *suppurative parotitis*, suppurative otitis and nephritis. In grave cases severe bedsores may develop.

Prognosis. Typhus runs a much more benign course in children than it

does in adults. Severe cases are rare (10-12 per cent), cases of moderate severity occurring more frequently; sometimes, especially in nurslings, the disease takes an abortive and mild course. Mortality is very low, actually close to zero (0-0.3 per cent).

Hygiene, care, treatment. The patient's room must be aired; the bed must be made carefully, the linens changed frequently, and the skin on the sacrum and back constantly examined because severe cases give rise to bedsores. In severe cases a rubber ring must be placed under the child and a cold compress applied to the head. Daily warm baths (35-37°C) prove quite effective. In cases with high fever the warm baths may be supplemented with cool packs.

The mouth must be thoroughly rinsed either with a 0.85 per cent sodium chloride solution, a 2 per cent boric acid solution or a 0.1 per cent potassium permanganate solution. The tongue and lips must be coated with vegetable oil. Swabbing the mouth is not recommended because it only injures the mucosa.

The patient must be given enough to drink and a *nourishing diet*; during the febrile period the food must be liquid or semiliquid. Dairy dishes, soups and broths, kissel and cereals are usually prescribed. If the child's condition is not serious, meat and fish are not contraindicated. The child must get vitamins—fruit juices, fish-liver oil, sweet cream, butter. During convalescence the patient must be given fish-liver oil. The child may be allowed out of bed two or three days after the drop in temperature. There is no need prescribing cardiacs. If such a need arises, the child is given a teaspoonful (dessert- or tablespoonful) of a 1 per cent caffeine solution, cordiamine or cardiazol.

The patient may be discharged from hospital five or six days after the drop in temperature.

Typhus is now very effectively treated with antibiotics (synthomycin, biomycin) which often arrest the course of the disease.

Prophylaxis. The main efforts must be directed toward exterminating the vector of the disease (the louse) and creating conditions under which the louse cannot multiply. *General improvement of sanitation and hygiene*, raising the cultural level of the population and health education of the population are the decisive factors. All forms of collective, properly organised life of the children are potent sanitation factors. In properly organised children's institutions infection, even if brought in, cannot spread and develop. If the disease breaks out among the adult population, special attention must be devoted to ridding the children of lice because children are easily infected and may often themselves serve as sources of the infection. Places frequented by many people (railway stations, baths, etc.) must be kept under special control.

The disinfection measures consist in extermination of the insects (disinsection). The underwear and clothing are put through dry-air or paraformaldehyde chambers. The underwear may be boiled or pressed with a hot iron. The interiors of buildings are disinfected by fumigation with sulphur. Preparation K (insecticide, $C_2S_4O_2/C_2H_5/2$) and soap K which contains this preparation are very effective insecticides. The typhus patient admitted to the hospital must be thoroughly washed and his hair must be clipped.

A method of specific prophylaxis (Krontovskaya, Mayevskaya, Pshenichnov) has recently been elaborated and tested in practice.

RELAPSING FEVER

Etiology. The causative agent of relapsing fever is a parasite of animal origin—a *spirochete* living in the patient's blood. The spirochete was discovered by Obermeier in 1868. It is a thin, very motile spiral with several curls; it is easily found in the patient's blood during an attack, in smears or in a heavy drop and a hanging drop with a narrow diaphragm, or by a dark-field examination; it stains well with aqueous fuchsin, methyl blue or Romanovsky-Giemsa stain.

During an attack the patient's blood contains a large number of spirochetes; during the apyretic period they disappear from the blood.

The infection is transmitted through the *body louse* which is the only vector of the relapsing fever causative agent. It has been established that the louse can infect a healthy person only 4-5 days after sucking the blood of the patient containing spirochetes, during which period the spirochetes in the organism of the louse go through a cycle of development and reproduction, disappearing from the intestinal tract of the louse and entering the tissues of its body. The infection is therefore transmitted not through the bite of the louse or through its excrements (as in typhus), but only when the louse is crushed on the body and the spirochetes enter the wounds and scratches on the skin.

The *epidemiology* of relapsing fever is very much like that of typhus. Relapsing fever is also a concomitant of war, famine and poor sanitation and hygiene. The two infections often occur simultaneously. The cause of their onset is the same; lack of soap, clothing, fuel and water, overcrowded and filthy housing and, especially, a low cultural level of the population, create conditions for the breeding of lice; as soon as disease appears the insects are infected en masse and in their turn infect the people and give rise to an epidemic.

Like the outbreaks of typhus, those of relapsing fever occur mainly in *autumn and winter*. The disease attacks older children who have greater contact with infected material. The incidence of relapsing fever in the U.S.S.R. has now been reduced practically to zero.

Pathological anatomy and pathogenesis. Sections show symptoms of degeneration of internal organs and considerable enlargement of the spleen; the latter usually exhibits large infarctions, not infrequently miliary necroses and abscesses. Abscesses caused by a bacillus which resembles the typhoid bacillus and enters from the intestinal tract may also be observed in other organs—kidneys and liver. Sometimes there are hemorrhages into internal organs and the adrenals. The intestinal tract is often affected with ulcerative colitis. The alternation of attacks and apyrexia are due to accumulation of antibodies in the blood followed by a new multiplication of the parasite. Immunity is apparently temporary. Recurrent cases are not infrequently observed soon after the first attack of the diseases.

Clinical findings. The *incubation period* averages 7-8 days, but may be 3-14 days. The onset of the disease is very rapid, sometimes sudden, the temperature rising to 39.5-40°C. In other, less frequent cases the disease may develop over a period of 2-3 days. At the same time the patient has intense headaches, often chills, nosebleed and nausea; vomiting and sometimes intestinal disorders are observed.

Subsequently a peculiar picture develops—alternation of *febrile attacks* and *apyretic periods*.

During the attacks the temperature varies in intensity—about 39-40° and symptoms of *general intoxication* with severe headaches, muscular and nervous pains in the legs and back, especially in the thighs and shanks, sacrum and back of the head, are observed; some cases are attended with delirium and *meningeal symptoms*.

Examination of the skin in some cases reveals herpes and transitory erythemas; the skin is dry and sometimes perceptibly icteric; the scleras are not infrequently slightly icteric. The tongue is coated with a white film ("milky" or "chalky" tongue); it is characteristic that despite high fever it remains moist and broad.

The intestinal symptoms are often characterised by diarrhea, sometimes with mucus and blood, but constipation is more usual.

Very early—from the very first days—the spleen begins to enlarge. It may enlarge considerably and protrude 2-3-4 digits from under the ribs. It is moderately dense. Sometimes it is painful to touch. In some cases (unlike typhoid fever and typhus) it may become very large. It contracts sharply during apyrexia and enlarges again during attacks.

During attacks the *appetite sharply diminishes* and the child loses a good deal of weight. The liver is moderately enlarged and sensitive to touch.

The *respiratory* symptoms consist in bronchitis and not infrequently nose-bleed.

At the height of attacks the *heart becomes enlarged*, the heart sounds not infrequently grow dull, and sometimes a *clear systolic murmur at the apex* appears; there is some drop in blood pressure and a diminished tension of the pulse. The pulse is quickened. There have been cases of sudden collapse with lethal results caused, as has been established by autopsy, by *hemorrhages into both adrenals*.

The urine sometimes contains albumin and almost always a great excess of urobilin.

During attacks the blood shows high leukocytosis (10,000-20,000) of a neutrophilic character, alternating with a normal leukocyte count during apyrexia, relative lymphocytosis and monocytosis; the erythrocyte sedimentation rate is high. The hemoglobin content of the blood and the erythrocyte count decrease rather sharply.

At the end of attacks the temperature drops critically, which is very characteristic of relapsing fever. The patient's general condition rapidly improves, and only weakness, malnutrition and anemia remain.

An attack lasts 3-6 days and apyrexia—5-9 days. The number of attacks varies—from 2-3 to 8 (severe cases). It is interesting to note that in the course of the disease the attacks grow shorter and apyrexia longer.

The duration of the disease is about two and a half to three weeks.

Complications. Degeneration of the heart muscle and myocarditis are very infrequent in children; endocarditis occurs as a rare exception. Renal symptoms consist in albuminuria. Iritis is a rare but very characteristic complication of relapsing fever.*

Serious affections of the joints are also rare. *Ulcerative colitis with mucus and blood* is a rather frequent occurrence. This complication greatly resem-

* In connection with the infectious process affection of the iris in children occurs mainly in two diseases—*meningococcal cerebrospinal meningitis* with manifestations of meningococcemia and in *relapsing fever*; iritis occurs very rarely in *rheumatism*, *tuberculosis* and *syphilis*.

bles bacillary dysentery but is apparently caused by the action of the spirochetes, as are also all the aforementioned complications.

Another series of complications is connected with penetration of *purulent coccal infection* into the organism; this includes pneumonia, empyema, nephritis, suppurative otitis, suppurative parotitis and purulent pericarditis. Multiple abscesses of the spleen, liver and kidneys in children have been described and are associated, as was demonstrated by Kulesha and Ivashintsev in the case of adults, with the entrance from the intestinal tract of a peculiar type of paratyphoid bacillus (*N-paratyphoid bacillus*).

The *prognosis* and severity of the forms of the disease depend on the peculiarities of the epidemic and the patient's general condition.

Mortality is very low and is conditioned mainly by the complications. In weak, emaciated children it may be considerably higher.

Care, hygiene and treatment. Care of the patient is subject to general rules (see *Typhus*). It is very important to devote special attention to adequate alimentation of the child. Relapsing fever causes considerable loss of weight and emaciation. The patient must be given enough vitamins.

Specific treatment. *Salvarsan* acts specifically on spirochetes. It was effectively used for the first time on a large number of patients by the Russian physician Iversen in 1909. Preparations of neosalvarsan, novarsan, etc., are administered intravenously in doses of 0.015 g per 1 kg of the patient's weight. To terminate the disease it is usually necessary to make two infusions: the first, at the height of the first attack and the second, on the 4th or 5th day of the first apyrexia. The entire requisite dose of 0.15 g for a child weighing 10 kg, or 0.2 g for a child weighing 12 kg is dissolved in 1-1.5 ml of an 8-10 per cent glucose solution and is injected into the ulnar vein.

Information on the effectiveness of penicillin in relapsing fever has been obtained in recent years. In cases of cardiac weakness caffeine is administered during attacks.

The *prophylaxis* is the same as in typhus.

INFLUENZA

Until very recently the term influenza implied an acute infectious disease characterised by catarrh of the upper respiratory tract and manifestations of general intoxication (elevated temperature, headache, indisposition, etc.).

However, more than 60 years ago the outstanding Russian pediatrician N. Filatov showed that the term influenza actually united two diseases differing clinically, epidemiologically and apparently etiologically.

In recent years this view of Filatov has been fully confirmed by microbiological and serological studies, as well as clinical and epidemiological observations. Two fundamentally different diseases are distinguished today (to be exact, two groups of diseases): epidemic influenza and seasonal (febrile) upper respiratory catarrh.

Epidemic influenza is very contagious; it conditions recurrent epidemic outbreaks regardless of the time of the year and climatic

conditions, and affects adults and children alike, producing quite severe forms of the disease which runs a peculiar course. These outbreaks may assume the nature of extensive epidemics (pandemics) which now and then involve many countries. Such epidemics have been observed at intervals of about 25-30 years.

Seasonal upper respiratory catarrh is less contagious and the infection is transmitted during closer contact; constantly occurring among the population it grows more intense in autumn, winter and early spring, but never affects such masses of people as does epidemic influenza; it very frequently affects children, especially very young ones.

Epidemic influenza has been known since the 16th century, a number of extensive epidemics striking, one after another, many European countries.

Two pandemics, well-studied and described, affected the greater part of the world in 1889-1890 and 1918-1919 and took a toll of millions of human lives.

The 1918-1919 epidemic outbreak was called the "Spanish disease" on the assumption (which proved wrong) that the disease started in Spain.

Weaker, although still substantial waves were observed in between the extensive epidemic outbreaks. These waves rolled over a number of countries in 1927, 1929, 1931, 1933 and later. The disease spread very rapidly, affecting mainly the adult population and older children.

Etiology. For a number of decades the Afanasiev-Pfeiffer bacillus was considered the causative organism of influenza. It is one of the minutest, nonmotile, Gram-negative organisms; it lives in media with admixtures of blood.

In recent years it has been demonstrated, however, that the causative agent of influenza is a pneumotropic virus whose two varieties have been studied, namely, virus A and virus B which differ in their antigenic pattern and therefore do not produce reciprocal immunity (i.e., influenza caused by virus A does not exclude the possibility of infection with virus B).*

The influenza virus is very unstable in the external environment and quickly disintegrates, especially, under the influence of sunlight and desiccation.

The diseases caused by the different types of virus cannot be differentiated by clinical signs. The blood serum of humans and

* It was recently demonstrated that, in addition to viruses A and B, viruses C and D are also causative agents of influenza.

animals who have survived an attack of influenza caused by virus A neutralises it but does not affect virus B and vice versa. The influenza virus is grown in tissue cultures containing Tyrode solution and embryonic tissues of guinea pigs or hens.

The site of primary localisation of the influenza virus is the mucosa of the nose, pharynx and upper respiratory tract.

In addition to the virus which produces pathological changes in the mucosa of the respiratory tract, an important part in the appearance of a number of complications in influenza is played by other microbes, especially the Afanasiev-Pfeiffer bacillus, as well as pneumococci and streptococci (so-called concomitants of the influenza virus).

Pathogenesis. The microbe is transmitted in droplets and apparently infects the organism through the respiratory tract; it is responsible for considerable phenomena of toxicosis, the nervous system, especially the vegetative, and the vascular apparatus being particularly affected. Hemorrhages, tissue necrosis (for example, of the alveolar walls) and considerable degeneration of the affected organs are quite frequent.

In addition to these, purulent and septic processes caused by secondary infection (diplo-streptococci, Afanasiev-Pfeiffer bacillus, etc.) are observed.

Influenza, especially its severe forms, creates conditions (even more favourable than does measles) for the spread of various secondary infections along the mucous membranes of the respiratory organs in virtue of the affection of the tracheobronchial tract, along the lymphatic vessels because of the deep affection of the interstitial tissue of the lungs (peribronchitis, interstitial pneumonia), and along the circulatory system owing to the considerable toxic affection of the vessels.

All these factors assume particular importance against the background of the general marked effect produced by the influenza virus on the nervous system and the changes caused by the virus in the reactivity of the organism (its sensibilisation).

An attack of viral influenza confers immunity for a period of 1-2 years.

Cases of influenza recurring after a relatively short time are accounted for by infection with different and possibly as yet undetermined types of viruses.

Clinical findings. The incubation period is 1-3 days. The onset is acute, without marked prodromal symptoms, with a rapid rise in temperature and general nervous symptoms. Intense headaches, mainly in the region of the forehead, orbits and temples, as well as hyperesthesia, are characteristic; children are irritable, cranky, sometimes apathetic; they have sleep and appetite disturbances

and are often delirious. Vomiting and nosebleed are frequent. The disease has various forms; in some cases nervous symptoms predominate (*toxic form*), in others affections of the respiratory tract and catarrhs of the mucous membranes are more clearly pronounced (*toxis-catarrhal form*). A *mixed form* is more frequently observed. Generally characteristic of viral influenza is the fact that during the first days of the disease with clearly marked symptoms of general intoxication the catarrhal symptoms are pushed into the background and become more distinct only within 2-3 days.

Examination reveals moderate catarrhs of the mucous membranes; on auscultation many dry and not infrequently moist rales scattered all through the lung, as well as mild emphysema, are noted. *Conjunctivitis* and *coryza* (unlike endemic influenza) are *feebly marked*. The vascular apparatus shows symptoms of weakness—a certain, rather early, dilatation of the heart with very characteristic symptoms of a *slow pulse* (bradycardia). Simultaneously *leukopenia* (5,000-2,000 leukocytes) with a relative lymphocytosis and low eosinophil count is often observed. The erythrocyte sedimentation rate is normal or somewhat faster, the blood pressure is low; sometimes there is a tendency to a liquid stool, profuse perspiration and frequent affection of the respiratory tract. All these symptoms denote affection of the parasympathetic division of the nervous system.

Severe cases are attended with nephrosis. As complications which sometimes develop very early, almost from the very first days of the disease, *pneumonia*, *pleuropneumonia* and *purulent pleurisy* are observed. Severe cases are attended with hemorrhagic pneumonia, hemoptysis, nosebleed and cerebral hemorrhages. Cases of *mortification* and *abscesses* of the lung, necrosis of bones and mucous membranes resulting from vascular thrombosis have been described.

Quite peculiar are the necrotic lesions of the upper respiratory tract—*larynx*, *trachea* and *bronchi*—which lead to severe croup with symptoms of *stenosis* that sometimes necessitates intubation or tracheotomy.

The temperature curve is very peculiar: often it is “two-horned” or resembles the outlines of a bell. The febrile period in uncomplicated influenza lasts 3-6 days.

The temperature drops both critically and lytically, and after the drop the patient may feel generally indisposed, jaded and weak for a week or more, and the longer, the older the child.

The process lasts an average of 1-2 weeks. Mortality depends on the form of the disease and varies with the different epidemics.

In addition to moderately severe forms there are also mild forms with a one day's rise in temperature.

Fortunately, children are rarely affected with hypertoxic forms of the disease. These forms run an extraordinarily stormy course with a picture of predominant affection of the nervous system and vascular apparatus, and sometimes end lethally during the very first two days.

There are as yet no reliable methods of early diagnosis of influenza. A laboratory diagnosis is made by administration of cultures from the nasopharynx of patients to susceptible animals or into a developing chick embryo for isolation of the virus. Of late an erythrocyte agglutination test (hemagglutination) is used for diagnosis; this test establishes the existence of the viral antigen.

A neutralisation reaction, complement fixation and retarded hemagglutination tests are used for diagnosing influenza.

One of the laboratory methods is a microscopic examination of smears from the nasal mucosa; characteristic of influenza is the presence of a large number of columnar epithelial cells.

Seasonal upper respiratory catarrh is one of the most widespread children's diseases, occurring everywhere and affecting children of all ages, but particularly frequently very young children. The disease incidence increases during the cold time of the year possibly because children stay less outdoors and therefore do not get enough fresh air, and because of greater contact with patients and carriers of bacilli. Seasonal catarrh may recur because it does not confer permanent immunity.

The question of the *etiology* of seasonal catarrh is not quite settled as yet. The disease is believed to be caused by the usual pathogenic microflora of the nasopharynx (pneumococcus, streptococcus, catarrhal micrococcus, influenza bacillus, etc.). There are experimental data indicating its viral etiology; this etiology has now been given definite confirmation.

The incubation period is short—from one to three days. The onset is characterised by general phenomena of intoxication—indisposition, headache, elevated temperature and development of catarrhs of the mucous membranes, i.e., coryza, cough and conjunctivitis. During this period seasonal catarrh very much resembles measles.

Examination of the child at the height of the process frequently reveals, in addition to the catarrhs of the mucous membranes, symptoms of laryngotracheobronchitis with a dry, often raucous cough and dry rales in the lungs. No appreciable disorders of the cardiac function are usually noted. Very young children often have

intestinal disorders (parenteral dyspepsia). The nervous symptoms include general irritability, hyperesthesia and muscular pains, restlessness and crankiness. Moderate neutrophil-type leukocytosis and sometimes leukopenia are observed in the blood.

The *course of the disease* varies. The process often terminates in 2-3 days without serious disorders; in other cases it lasts 5-7 days with irregular temperature which drops lytically. In some cases the disease may be prolonged for several weeks.

After an attack of even mild influenza the child does not recover at once—irritability, nervous disorders, poor sleep and disturbed appetite persist for a long time. During influenza and immediately after it the child very easily contracts other diseases because of lowered resistance.

Complications. *Respiratory* complications are the most frequent. So-called *false croup*—stenotic laryngitis—is often observed during influenza; this disease not infrequently develops from the very first days of influenza, the child's parents and other people surrounding the child being greatly frightened by the abrupt appearance of respiratory difficulties. The process begins, as usual, with slight indisposition, gradual moderate rise in temperature (37.8-38°), coryza and mild cough. Suddenly, often in the middle of the night, the cough becomes dry and hoarse, and a picture of difficult stertorous respiration accompanied by great excitement and restlessness develops. In the fauces there is nothing except hyperemia; there are no membranes and the voice is usually retained. The process in the larynx is characterised merely by hyperemia and edema without a fibrinous membrane as in diphtheria.

This distinguishes *false croup* from *true diphtheritic croup* in which the phenomena do not develop so rapidly, there are no catarrhs of the mucosa, while examination of the fauces, nasopharynx, nose or epiglottis with a mirror often shows fibrinous membranes. A similar fibrinous exudate is also observed in the larynx during diphtheria.

False croup is not dangerous and usually soon terminates. A considerable part in its genesis is apparently played by laryngeal spasm caused by hyperemia and edema.

The most serious complication is *pneumonia*. It is most frequently *lobular*, in the form of small foci disseminated through both lungs, and is very often accompanied by bronchitis and peribronchitis, which is discovered during section and is clinically manifested in a prolonged febrile process and, subsequently, sometimes in formation of *bronchiectasis*, as in measles and whooping cough. During pneumonia and following it secondary *empyemas* may develop. Pneumonia usually runs a severe course and is most frequently

the cause of death in influenza. During influenza the *middle ear* is often affected with resultant simple or suppurative otitis. *Shooting pains in the ear* at the onset of such otitis are a very typical symptom. Not infrequently pyelitis develops in the course of influenza, especially in very young children.

Meningitis and meningoencephalitis caused by the Afanasiev-Pfeiffer bacillus are observed relatively rarely.

Prognosis. Respiratory catarrh is an extraordinarily widespread disease. It often complicates and aggravates the course of other diseases. Frequently producing respiratory complications, often ending in pneumonia, causing pyelitis and reducing the patient's resistance to other diseases, tuberculosis in particular, seasonal catarrh is a very dangerous disease, especially for young children.

Treatment. *Care, hygiene and diet* are very important. For fear of exposing the child to cold some families for a long time deprive the child affected with influenza of fresh air and baths. During influenza these measures must, on the contrary, be extensively used. The patient's room must be constantly aired. In warm weather the patient *may be kept outdoors* or in the room with windows wide open all day.

Daily warm and hot general baths or hot foot baths greatly relieve the nervous symptoms and muscular pains. In cases of pneumonia in nurslings and very young children *mustard packs* or *circular mustard plasters* and hot baths (38-39°C) prove very effective.

Per os the patient is administered a 2 per cent sodium bicarbonate solution or alkaline waters (borzhomi, Izhevsk water). Expectorants are not recommended because they intensify the cough which is undesirable in cases of peribronchitis and possible bronchiectasis. A proper diet is very important; for nurslings it is important to retain human milk and full-value nutrient mixtures, while older children do not have to be deprived of meat. It is necessary to administer vitamins (fish-liver oil, fruit juices, egg yolks). During dyspepsia children past six months of age are given kefir. In cases of otitis warm oil is instilled into the ear and hot compresses are applied; for intense pains older children may be given 0.1-0.2 g of aspirin two or three times a day. During attacks of false croup—hot general baths or hot foot baths (with or without mustard); hot drinks and bromides *per os*, and inhalation of hot vapours.

The same measures are used in the treatment of viral influenza. In view of frequent cardiac disorders the patients are administered

a 1 per cent caffeine solution, cordiamine and cardiazol per os; in acute cases—cardiacs subcutaneously.

Complications are treated according to general rules. Severe symptoms of stenosis sometimes necessitate a tracheotomy, although it cannot be very effective owing to the rapid development of pneumonia and symptoms of serious general intoxication. Pneumonia is treated as usual. Sulfa drugs are of little avail in pneumonia caused by the Afanasiev-Pfeiffer bacillus. Combined sulfazole (sulfamethylthiazole) and penicillin treatment is more effective. Diathermy and ultrashort waves are indicated.

Prophylaxis. Proper organisation of the child's, especially very young child's, everyday life is of paramount prophylactic importance. The child must be given plenty of air and light, for which purpose it should be kept outdoors as long as possible and the windows in his room should be open during the warm time of the year. Systematic airing of the living quarters and long walks in the open are particularly important in the autumn and winter; cold water rubdowns in the morning are recommended for older children and bathing for very young children.

The experience of properly organised children's institutions (nurseries, children's homes, kindergartens, summer camps, etc.) shows that these procedures strengthen and harden the child's organism and make it less susceptible to infections, influenza in particular. As for personal hygiene, it is necessary to teach the child to keep his hands clean and take care of his oral cavity. Adults must not shake hands with children or kiss them.

If the mother or person looking after the child has contracted influenza, she must be instructed carefully to observe the rules of prophylaxis, i.e., wash her hands frequently and wear a gauze mask covering the mouth and nose during contact with the child. This applies particularly to nursing women. As a general rule, children must be kept away from persons who are affected with coryza, or cough, or who are recovering from any infection.

In cases of influenza breaking out in hospital wards the patients are placed in cubicles, while the ward is thoroughly aerated and a number of sanitary measures are carried out.

Research aimed at elaborating specific influenzal prophylaxis is now being insistently conducted.

Encouraging results have been obtained from subcutaneous administration of a concentrated polyvalent vaccine prepared from an allantoic culture of viruses A and B. Smorodintsev, a Soviet scientist, recommends administration of a pulverised antiserum into the nose for prophylactic (as well as therapeutic) purposes.

The trouble with this method is that it does not confer stable immunity.

Yermolyeva and Valedinskaya, well-known Soviet researchers, have recently proposed *ecmolin* (an antibiotic preparation of animal origin) for prevention (as well as treatment) of influenza; the preparation is instilled or sprayed into the nose.

WHOOPIING COUGH

Etiology. The causative agent of whooping cough is a bacillus discovered by Bordet and Gengou in 1906. The microbe is isolated from the patient's sputum and is a short, nonmotile, Gram-negative bacillus. The bacillus is cultivated under aerobic conditions in a special medium containing blood (blood-potato-glycerin agar); its harmful effect is due to substances contained in its body.

Epidemiology. Man is very susceptible to whooping cough, the disease affecting all ages, especially children from one to five years old; nurslings are affected by whooping cough more often than by any other infectious disease.

More than 60 per cent of the morbidity and 95 per cent of mortality from whooping cough occur during the first three years of life, the highest lethality being observed during the first year of life. The lower disease incidence among children during the first years and months of life is accounted for by the fewer possibilities of their infection and partly by their passive immunity. However, there have been cases of whooping cough in children a few days old, who contracted the disease from their mothers.

The causative agent settles in the respiratory tract and infection is transferred by means of droplets of moisture expelled by crying, sneezing and coughing. The infection is not transmitted through third persons or things because outside the organism the microbe is not very viable. The radius of its dissemination is small and with timely and proper isolation of whooping cough patients the infection very seldom spreads. Accidental and brief association with a whooping cough patient may undoubtedly result in transfer of the infection, but, unlike measles, infection usually occurs as a result of longer and closer contact. The patient becomes contagious from the very first days of the disease when it is frequently impossible to diagnose, which increases the danger of spreading it. The disease is the most contagious at the end of the catarrhal and the height of the convulsive periods, then the contagiousness gradually diminishes and ceases about 30 days

after the beginning of the coughing attacks and 40 days after the onset of the disease. At this time whooping cough bacilli are no longer excreted even if the coughing continues.

Poor living conditions (congestion, failure to observe the rules of sanitation and hygiene, lack of fresh air, deficient diet) foster morbidity and aggravate the course of the disease. Violation of the established regimen and the rules of isolation in children's institutions may easily stimulate outbreaks of whooping cough among young children.

In large communities whooping cough occurs endemically; the epidemic waves come at intervals of 2-4 years. No regular distribution of morbidity according to seasons is observed. Of considerable epidemiological importance are patients with inconspicuous forms of the disease which most frequently affect older children and sometimes adults; these forms are also observed in very young children. Recent observations confirmed by bacteriological and serological studies have established that in massive whooping cough foci such inconspicuous forms (without paroxysmal coughing) occur in a large percentage of cases (up to 30 per cent). This fact must be taken into account in carrying out prophylactic measures. An attack of the disease confers lifelong immunity.

Pathogenesis and pathological anatomy. Whooping cough is a general disease with the microbial poisons (endotoxins) producing a peculiar effect on the nervous system and mucous membranes of the *respiratory tract* causing an inflammation of these membranes.

In addition to the selective specific action of the whooping cough poison on the nervous and respiratory systems, a significant part in the genesis of the clinical manifestations is also played by secondary, nonspecific factors. For example, the mechanism of development of paroxysmal coughing is as follows: stimulation of the receptors of the mucosa of the respiratory tract (larynx, trachea, bronchi) by the whooping cough microbe and its toxin gives rise to a focus of excitation in the corresponding parts (respiratory and cough centres) of the brain. This focus of excitation is characterised by features of a dominant focus.

For this reason any stimulation—a psychic factor, examination of the throat, addition of a secondary infection, for example, influenza, measles, etc.—easily evokes a response from this focus and may cause the attacks of coughing to be intensified or prolonged; contrariwise, this focus may be suppressed by creation of other foci of excitation (there have been cases in which the attacks of coughing ceased or diminished when the patient's surroundings changed or the patient was engaged in an interesting game or was otherwise distracted).

Causing disturbances in the respiratory rhythm and a prolonged cessation of inspirations paroxysmal coughing leads to disorders of pulmonary ventilation and oxygen deficiency (hypoxia and hypoxemia). Disturbances in the oxygen exchange and circulatory disorders in the central nervous system

cause convulsions and other nervous manifestations, especially in very young children who are particularly sensitive to oxygen deficiency.

The whooping cough toxin also affects the vascular system, which is attested by the observed capillary spasm and paresis; it increases capillary permeability, causes a change in blood pressure, affects the metabolic processes (the vitamin balance in particular) and considerably decreases the immunobiological resistance of the organism.

The pathoanatomical picture is characterised by a tendency to development of inflammation not only in the walls of the bronchi but also around them—*peribronchitis*—which not infrequently leads to intense development of connective tissue in the lung (*pulmonary cirrhosis*). Pneumonia which often attends whooping cough is of a diffuse character (lobular pneumonia); in connection with the aforementioned peribronchitis pneumonia is often followed by dilatation of the bronchi (bronchiectasis).

Clinical findings. The incubation period is usually 5-8 days, seldom shorter—from two to four days—or longer—up to 21 days. Three periods are distinguished in the course of the disease: *catarrhal*, *spasmodic* (paroxysmal coughing) and *resolution*.

Whooping cough begins like ordinary coughing, sometimes with a rise in temperature and sometimes without.

Coryza and mild conjunctivitis are not infrequently observed in very young children; pharyngitis and laryngitis occur less frequently. At this period whooping cough is at first easily confused with influenza and upper respiratory catarrh. However, in the latter cases the signs of catarrh (coryza, conjunctivitis, angina) are much more strongly pronounced and the temperature is higher.

Subsequently the coughing becomes increasingly more stubborn and persistent, appears at indefinite intervals and, what is particularly characteristic, *at night*. At this time, however, the patient's general condition undergoes little change. In this type of coughing drugs are scarcely effective. The first, catarrhal period lasts from one to two weeks, but may vary from several days to three and sometimes four weeks. As a general rule, it is the shorter, the younger the child. This period is followed by the second, spasmodic stage. In the second or third week the coughing assumes the character of paroxysms. The coughing attacks occur suddenly, the cough impulses follow each other rapidly and give the child no chance to catch his breath. The child is said to have a "coughing fit". He looks frightened, turns blue in the face and his respiration is suspended; he opens his mouth wide, sticks out his tongue, jumps up and catches at surrounding objects; the attack produces a distressing impression. Then the child takes a deep breath, which is sometimes a loud whooping inspiration due to *spasm* of the glottis. This is again followed by a coughing fit and a deep, whooping inspiration; the whole picture may recur several

times. The child turns blue and his face purple. Finally the attack ceases and the child coughs up a small amount of viscous phlegm; not infrequently the attack ends in vomiting. Such an attack is for some time followed by a period of ease: the child plays and feels good, but then everything begins all over again.

In the beginning of the disease such attacks are very short and infrequent, but as the disease develops, especially its severe forms, they occur very frequently, give the patient no rest day or night and greatly exhaust him.

The other symptoms of whooping cough observed at the height of the process include a peculiar *puffiness* of the face, edema of the lids and swelling of the lips, greyish skin with a cyanotic tint, a general pastiness (especially of the hands and feet), often hemorrhages into the conjunctiva, sclera, the skin of the lids and face, and nosebleed.

Very often, in severe forms of whooping cough a small *ulcer* covered with a whitish film appears under the tongue, on the frenulum or the tip of the tongue; the ulcer is a result of the tongue's rubbing against the sharp incisors. It is very characteristic that in uncomplicated cases of whooping cough, even when there are intense coughing attacks, there are no appreciable changes in the lungs. Only an overdistension of the lungs (emphysema) (for which reason it is difficult to determine cardiac dullness) and dry rales can be observed. Roentgenological examination reveals in most whooping cough patients intensified pulmonary contours and marked cords in the region of the roots of the lungs. These changes are most clearly pronounced at the height of the spasmodic period, but sometimes also persist after clinical recovery.

Considerable leukocytosis and lymphocytosis are discovered in the *blood* in the first or second week of the disease; the erythrocyte sedimentation rate is normal or somewhat slower. In the early periods of the disease the *urine* has high specific gravity and contains a large amount of uric acid.

These changes in the blood and urine, stubborn cough, normal or slightly elevated temperature and absence of phthisical data in the lungs warrant suspicion of whooping cough.

The spasmodic period lasts from 2-3 weeks to two months, averaging 4-5 weeks. Then the attacks diminish in intensity and frequency, and the disease imperceptibly enters the third period—the resolution period in which the cough loses its paroxysmal character. Uncomplicated whooping cough lasts an average of 1.5-2 months, but may persist for three months. Even later, however, in case of any respiratory disease (influenza) and under the

influence of physical and mental stimuli the cough may resume the character of attacks. But, as was already mentioned, towards the 40th day after the onset of the disease the patient is no longer contagious and the remaining tendency to cough is a manifestation of a reflexly evoked trace reaction. The clinical forms of whooping cough may vary in severity and course. These forms are classified as mild, moderately severe and severe, according to the frequency and intensity of the attacks and the duration of the spasmodic stage.

Mild cases sometimes run an atypical course and may be overlooked. In these cases the spasmodic period is short and weak. The attacks are rare and not intense. There may be no vomiting. These cases are difficult to diagnose. Whooping cough is indicated by persistent coughing and its periodicity, prevalence of nocturnal attacks, tenseness and redness of the face during coughing, restlessness, discharge of phlegm at the end of coughing, absence of special pulmonary symptoms (except emphysema), and the above-described changes in the blood and urine; it is also important to take into consideration the epidemiological data.

Bacteriological and serological methods may be used for diagnosing atypical forms of whooping cough which show but few symptoms. The material for cultures is taken by means of cough plates, an open Petri dish with a special nutrient medium being brought to within 6-10 cm of the coughing child's mouth. By this method it is possible to obtain positive results in 60-70 per cent of the cases even during the catarrhal period. Valuable data are obtained by means of the complement fixation test. To perform this test, it is enough to take 0.3-0.5 ml of blood from a finger. A positive reaction is obtained in the second week of the spasmodic period, and in inconspicuous forms—from the third or fourth week after the onset of the disease (cough). This test is important not only for the diagnosis of an individual case, but also for retrospective detection of an epidemic focus of whooping cough.

In *moderately severe cases* the attacks number 20-30 a day; all periods develop as usual. The disease lasts from five to six weeks.

In *severe cases* attacks occur very often (40-50 a day and more), each attack being long and intense. Malnutrition, sleep and nervous disturbances, and sometimes prolonged fever are observed. Complications are frequent.

The course of whooping cough in very young children (especially in the first year of life) has a number of peculiarities: the incubation and catarrhal periods are often short, the coughing attacks are peculiar, less frequently end in vomiting and are often

accompanied by respiratory arrest, asphyxia and convulsions. All this determines the greater severity of the disease and the frequency of complications, pneumonia in the first place.

Complications. The most frequent *respiratory* complication is pneumonia. It occurs more often in very young children, up to three years of age, especially in those suffering from rickets and hypotrophy. It is a disseminated, usually bilateral process of a microfocal character (lobular pneumonia). The foci merge so that pneumonia may involve considerable portions of the lungs.

Pneumonia usually develops in the second or third week of the spasmodic period and in a number of cases runs a sluggish course without appreciable rises in temperature; in other cases it develops rapidly, with high fever and toxicosis against the background of a generally grave condition. In the former cases the changes caused in the pulmonary tissue by whooping cough apparently play the most important part, whereas in the latter cases additional secondary infections are the main factor. Characteristic of pertussal pneumonia is the extensive affection of the bronchial system not infrequently involving *bronchitis* and *peribronchitis* and subsequently leading to *brinchiectasis*, shrivelling of pulmonary tissue (pulmonary cirrhosis) and complication with purulent *pleurisy*. Pertussal pneumonia is very dangerous to life and is most frequently the cause of death or protracted chronic processes; it also serves as an impetus to aggravation of tuberculosis. The other, less frequent complications include penetration of air into the pleural cavity due to breaks in the bronchial wall—*pneumothorax*—and into subcutaneous tissue—*subcutaneous emphysema*.

The digestive symptoms, especially in young children, are characterised by diarrheas.

The *nervous* complications are not infrequently manifested in *convulsions* (more often in young children), *paralyses* and, now and then, *encephalitis*. These complications may sometimes serve to disturb the subsequent proper mental development of the child. Intense attacks of whooping cough sometimes result in urinary incontinence and rectal prolapse.

Hemorrhages into the skin and mucous membranes (nosebleed) and, from time to time, *otitis* and *nephritis* are observed.

The *prognosis* is in large measure connected with age. For older children whooping cough is not very dangerous. For very young children (nurslings affected with rickets, weak and malnourished children) the disease is very dangerous; it often leads

to pneumonia and may end in death. Children affected with spasmophilia easily develop *convulsions* or *laryngospasm*. The disease is quite dangerous for *tuberculous children* because of the possibility of aggravating the process and developing *miliary forms* (tuberculous meningitis), aggravation of dysentery suffered by the child shortly before whooping cough is often observed.

Mortality (in hospitals) among very young children reaches 10 per cent, among nurslings—25 per cent and higher.

Treatment and care. One of the basic and most important requirements of proper treatment is to give the whooping cough patient as much fresh air as possible. It is well known that out in the open the attacks are less frequent and weaker, while complications, especially pneumonia and its consequences, occur much more rarely. Whenever possible the whooping cough patient must be kept outdoors both summer and winter. In winter and in any cold weather the child must be dressed warmly.

In severe cases with complications bedridden children are even in greater need of fresh air, and the *windows in the patient's room must therefore be always open*; the child needs fresh air particularly at night, when the coughing attacks are the most distressing. The child may be kept warm by hot-water bottles and warm underwear. Fresh air is especially important for young children. During the cold time of the year the patient's room must be constantly aired through open windows or transoms. The room must be kept clean, the cleaning being done with a moist rag. The whooping cough patient must not be allowed any contact with other patients, especially *influenza* cases. The addition of influenza to whooping cough facilitates development of pneumonia and renders the course of whooping cough more severe.

Since whooping cough attacks are intensified by excitement and vigorous movements, children who are not confined to bed must be prohibited noisy games and must be provided a regular, comfortable regimen with educational work aimed at raising their spirits and occupying their minds. The importance of this follows from what was said above concerning the pathogenesis of whooping cough.

The *diet* must be adequate with enough vitamins, especially vitamin C which favours better assimilation of oxygen. In severe cases patients must be given liquid and semiliquid food in small portions. In cases of vomiting it is sometimes necessary to cool the food and give it at more frequent intervals. In cases of frequent vomiting the child must be given the rest of his food after the attack and the vomiting.

Systematic warm baths (37-38°C) every other day are very important.

Medication. There are no specific drugs which terminate the attacks, although many have been proposed. A 1-2 per cent sodium bromide solution may be administered in severer cases. Narcotics do not help and therefore must not be administered, especially in cases of young children. Nor must expectorants be prescribed because they may intensify the cough impulses and foster development of bronchiectasis.

Treatment of whooping-cough with a specific vaccine has not gained wide currency since it is not sufficiently effective.

Some data obtained in recent years show that whooping cough can be effectively treated with antibiotics—streptomycin, synthomycin, levomycetin and biomycin. The sooner one of these antibiotics is used, the better the results. The antibiotics are administered in generally accepted doses: streptomycin in a daily dose of 25,000-30,000 u per 1 kg of the patient's weight and divided into two or three intramuscular injections; synthomycin in doses of 0.08 g, levomycetin—0.04 g and biomycin—25,000 u per 1 kg of the patient's weight per day; the latter three preparations are given per os in four portions. The course of antibiotic treatment averages 8-10-12 days.

Complications are treated by the usual methods.

The treatment of *pneumonia* is but again based on giving the patient as much fresh air as possible. In cases of diffuse lobular pneumonia the patients are administered mustard packs, mustard plasters and hot baths. Compresses are inadvisable because they hinder respiration. The patients may be kept warm by hot-water bottles.

If sulfa drugs given in the same doses as in other pneumonia cases prove ineffective, penicillin is simultaneously administered.

In cases of respiratory arrest in very young children it is necessary to administer artificial respiration, ensure an inflow of fresh air, give oxygen, inject 1 per cent lobeline or cytitone, administer cardiacs and, in attacks of convulsions, make a chloral hydrate enema.

According to some data, diathermy and ultrashort waves produce a favourable effect.

Prophylaxis. Specific vaccination. Inoculations with a pertussis vaccine are made to children up to five years of age never before affected with whooping cough and immunised against diphtheria. Children past five years of age are given inoculations only on epidemic indications. The vaccine is administered in three stages

in 1-ml doses with intervals of 3-4 weeks. For revaccination against whooping cough a pertussis-diphtheria vaccine is administered at the periods set for revaccination against diphtheria. Children never before affected with whooping cough and as yet not immunised against diphtheria are immunised at the age of 5-6 months with a pertussis-diphtheria vaccine (see *Prophylaxis of Diphtheria*).

Passive immunisation. Many authors recommend administration of the blood or serum of adults to children who have had contact with whooping cough patients. The dose is 30-50 ml.

Passive immunisation is prophylactically ineffective and yet, according to available data, it considerably mitigates the course of the disease and aids in reducing mortality even among the most vulnerable group of children under one year of age. This method is therefore indicated in cases of weak children, especially very young ones, affected with rickets or tuberculosis and in danger of contracting whooping cough after contact with whooping cough patients. Gamma globulin may be recommended for this purpose with antimeasles or hyperimmune serum (this serum is obtained by active immunisation of donors who have survived an attack of whooping cough).

General prophylactic measures. General sanitary and antiepidemic measures play the main part in the prophylaxis of whooping cough. *Early isolation* of the patient is very important. Every case of suspicious stubborn coughing, especially in children's collectives, must therefore be seriously attended to and the child must be isolated.

Association of the patient with healthy children is allowed six weeks after the onset of the disease or 30 days after the beginning of paroxysmal coughing.

Children who have had contact with a whooping cough patient must be kept under thorough medical observation for three weeks after the end of the contact. If these children begin to cough, they must be separated from the healthy children until ascertainment of the diagnosis.

Children up to ten years of age who never had whooping cough but had contact with whooping cough patients are forbidden admittance to children's institutions for 21 days after the end of their contact with whooping cough patients and, if their contact continues, for 40 days after the onset of the disease or 30 days after the beginning of paroxysmal coughing. Children past ten years of age and adults are admitted to children's institutions, but are kept under medical observation for 21 days.

In cases of appearance of whooping cough in children's institutions the practice of organising differentiated groups, i.e., isolation of whooping cough and whooping cough suspicious groups from healthy children, has justified itself.

MUMPS (EPIDEMIC PAROTITIS)

Etiology. Mumps is caused by a filtrable virus.

Epidemiology. This disease affects mainly children from five to fifteen years of age; in nurslings it occurs rarely. Adults contract it for the most part between 18 and 30 years of age. As a rule, mumps does not cause extensive epidemics, but may produce a number of cases in an institution in which there are poor sanitary conditions and close contact. The causative agent enters through the mucous membranes of the fauces and pharynx, through direct contact or droplet infection; the infection is not transmitted either through things or third persons. The lower mumps incidence (compared with measles, whooping cough, chickenpox) is apparently due to the fewer chances of transmitting the infection (the virus is in the saliva and there is no coughing) and the lower susceptibility of man to this infection. The patient is contagious from the first hours after onset of the disease and perhaps even several days before the glands appear to be swollen; the contagion persists for 2-3 weeks after the onset of the disease.

Pathogenesis. Mumps is a disease of the whole organism with a characteristic localisation of the process which affects the following parts: a) the system of salivary glands (parotid, submaxillary and sublingual), 2) gonads (ovaries and testes) and sometimes other glands (thyroid, pancreas), and 3) nervous system and internal ear. That this is a general process and not a local affection of the parotid gland is attested, in addition to the multiple localisation of the process, by the peculiar reaction of the blood and the hemopoietic apparatus, the cyclic nature of the course of the disease, relapses, changes in the cerebrospinal fluid (lymphocytic reaction), cerebral disorders and, lastly, lifelong immunity.

Clinical findings. The incubation period is 11-21 days, more often 18 days. The disease not infrequently sets in with general symptoms—indisposition, headache and sometimes nosebleed. In severe cases there may be vomiting, convulsions and meningeal phenomena. The temperature may rise very high—sometimes to 40°.

In other cases these general symptoms are feebly marked and the process is immediately, usually on the first day, less frequently on the second, and still less frequently on the third day mani-

fested in local phenomena. The pains begin on mastication and on opening the mouth, and are soon followed by a peculiar, slightly painful pasty swelling and edema in front of the auricle and below the ear flap, corresponding to the location of the parotid gland. The swelling may spread considerably both upward and downward. The skin over the swollen gland is glossy but does not turn red; no fluctuations are observed. In the beginning, and not infrequently all through the process, only one side, most often the left, is usually affected. In other cases the disease simultaneously affects both glands.

In some cases the tissue over the affected gland is extremely edematous and changes the facial expression so much that the mumps patient looks as if he were grimacing (the original meaning of the word mumps was "grimace").

Examination of the oral cavity not infrequently reveals an affection of the mucous membrane of the cheek at the orifice of the parotid duct or an edematous roll of mucosa, the orifice appearing as a bluish-red point.

There are no special changes in the organs, the tongue is coated moderately, salivation is sometimes affected, i.e., it is either decreased or increased. The lymph nodes are not involved. The blood shows leukocytosis (not in all cases, however), lymphocytosis and monocytosis. Temporary albuminuria is often observed.

All symptoms reach maximum intensity on the fourth or fifth day after which the morbid manifestations gradually diminish; the temperature drops lytically and everything ends by the eighth or tenth day if no other glands—the parotid gland on the other side (if the process was unilateral) and the submaxillary glands on one or both sides—are involved.

If the disease involves only the submaxillary glands from the very outset, it may run an atypical course and may fail to be diagnosed since in such cases it is characterised by considerable edema of the anterior part of the neck simulating the edema of severe toxic diphtheria.

Careful examination of the fauces and palpation of slightly painful swollen glands resembling spherical resilient bodies in the edematous connective tissue ascertain the diagnosis. In the absence of epidemiological data a primary affection of the submaxillary glands in mumps may be confused with periostitis of the lower jaw and acute lymphadenitis. The results of palpation show that in periostitis the most painful point of the swelling—its middle—is of very dense consistency and is closely connected with the bone; the skin over the swelling soon becomes red and glossy;

the diagnosis is also facilitated by the finding of carious teeth which are not infrequently a concomitant of periostitis.

Acute lymphadenitis is usually connected with simultaneous affections of the mucous membranes of the mouth, fauces and nose, the glands being more painful and dense, and no edema of the cervical connective tissue, as in the afore-described mumps, being observed.

Complications. In the overwhelming majority of cases mumps runs a favourable course without any complications.

Complications occur only in older children. Those caused by the action of the infective principle include inflammation of the testis (orchitis) and ovaries. *Orchitis* usually begins during the drop in temperature; the testis becomes swollen and painful, and the temperature rises again. The process lasts 7-10 days and usually ends well, only rarely causing atrophy of the organ. Inflammations of the ovaries are observed closer to the period of sexual maturation. The course is usually favourable.

Mastitis, pancreatitis and inflammation of the thyroid gland are infrequent complications. *Meningitis* and *meningoencephalitis*, usually running a benign course, are not so rare.

The knowledge of this fact is very important in making a differential diagnosis of meningitis of other etiology, especially tuberculous meningitis.

Peripheral neuritis and affections of the internal ear (labyrinthitis) with resultant deafness of one or both ears have been described.

Nephritis, serous affections of the joints and endocarditis are very rare.

Complications caused by secondary coccal infection—bronchitis, pneumonia, suppurative otitis—are sometimes observed.

Relapses. Sometimes the process, which seems to have terminated, suddenly breaks out again within two or three weeks and not infrequently affects the same gland as in the first attack; the course is shorter.

The *prognosis* is favourable. Lethal cases occur as a rare exception.

The *treatment* is symptomatic. Confinement to bed and a diet consisting of easily assimilable and chewable food, i.e., liquid and semiliquid. It is important to take care of the oral cavity by rinsing it with a 0.85 per cent sodium chloride solution or a boric acid solution. Application of all forms of heat to the affected gland—hot-water bottles, compresses or mere dry cotton bandages, and inunction of camphor oil.

In cases of orchitis and inflammation of the ovaries—heat and rest. Other complications are treated according to general rules.

Prophylaxis. According to the rules in effect until 1957, isolation of parotitis patients terminated 21 days after onset of the disease. Recent research has shown that the period of isolation can be reduced to nine days, the principal consideration being cessation of the acute manifestations of the disease.

A method of active immunisation against parotitis with a dry attenuated parotitis vaccine (A. Smorodnitseva) administered intracutaneously has now been elaborated.

Children who have had contact with mumps patients are also subjected to a 21-day quarantine beginning from the moment of contact; during the first ten days they may be admitted to children's institutions. The disinfection is general, consisting of airing, cleaning and washing. The child falling ill in a children's institution or hospital must be isolated in a separate ward or cubicle and ensured the usual individual care.

SERUM SICKNESS

The serum sickness is an allergic disease resulting from parenteral administration of foreign protein into the human organism.

The sickness is caused by injections of various serums for therapeutic or prophylactic purposes. Horse serum is usually employed.

Clinical findings. The time the signs of serum sickness appear, their duration and intensity vary and depend on the number of times the serum was introduced.

During the *first injection the incubation period* is 7-12 days, and more seldom 15-20 days; no manifestations are observed during the incubation period.

Symptoms may develop from the seventh day on and may consist of nervous and vascular phenomena, changes in the skin, lymph nodes, joints and blood. The most frequent and earliest symptom is an irregular rise in temperature which lasts from several days to three weeks. In some cases the serum sickness may not be attended with pyrexia. In the course of the serum sickness there may be remissions when the symptoms disappear and then reappear (relapses).

The manifestations on the part of the central nervous system consist in general jadedness, low spirits, irritability, headache and often vomiting; the changes in the vegetative nervous system are characterised by increased parasymphathetic tone (vagotonia).

The typical cardiovascular manifestations are a faster and somewhat weaker pulse (the dimensions of the heart remaining in most cases unaffected) and, as a rule, lower blood pressure.*

A cyanotic tint in the eruption and cyanosis of the lips and limbs are observed in more serious cases.

The stool is generally liquid and frequent; sometimes it even contains blood. Albumin appears in the urine (albuminuria).

At the height of the process the blood shows leukopenia and a decreased neutrophil count; the erythrocyte sedimentation rate at first drops and then rises; the thrombocyte count diminishes.

An acute dilatation of the lungs (emphysema) is quite often observed as a manifestation of increased vagal tone; the patient sometimes coughs, sneezes and exhibits symptoms of bronchitis.

The most constant and significant changes are those in the skin and the subcutaneous tissue in the form of various eruptions and edema. The eruptions are most frequently exudative, resembling urticaria or those in measles, rubella and sometimes scarlet fever. In some cases the disease is attended with hemorrhages of varying intensity.

The eruptions appear first at the site of injection and then may spread all over the body. *Itching* is a characteristic feature of the eruptions.

Exudative eruptions are often accompanied by local and general edema, especially frequently on the face and limbs; the edema is not due to renal affection, but is caused by retention of water in the tissues in virtue of the changes in their physicochemical properties.

It is also characteristic that the eruptions may change in appearance and intensity in the course of a day or even several hours and that they are generally conspicuous for their rather considerable polymorphism.

Swelling of the lymph nodes, beginning at the site of injection, i.e., usually at the inguinal glands, and spreading to other peripheral glands, is also typical and occurs more frequently and earlier than all other symptoms. This enlargement of the lymph nodes may be the only symptom of a latent or feebly marked serum sickness and constitutes its diagnostic importance. The *joints* not infrequently become swollen and painful.

The picture changes during repeated injections of the serum if Besredka's method was not used: 1) the serum sickness is ob-

* A faster pulse rate with a simultaneous drop in blood pressure is a very characteristic sign of the serum sickness and helps to diagnose it in vague cases. •

served much more frequently (in 90 per cent of the cases instead of the 30-60 per cent of those administered the serum for the first time); 2) the sickness sets in much sooner, sometimes immediately after the injection, and takes a much graver course.

An *immediate* (general and local) and an *accelerated* reaction is distinguished.

In cases of immediate local reaction the injection is followed almost at once (without the usual 7-12-day incubation) by edema of the tissue and redness at the site of injection; this is sometimes very painful, so that the picture simulates an acute inflammation (like an incipient phlegmon or erysipelas); in these cases the temperature rises quite high and the general symptom complex of the serum sickness rapidly develops. The clearest manifestations of an immediate local reaction are characterised by Arthus' phenomenon—a rapidly developing aseptic anaphylactic* necrosis at the site of the serum injection, which leads to mortification of the skin and subcutaneous tissue with subsequent formation of a slowly granulating ulcer.

The *immediate general reaction* is manifested in a rapid (during the first 6-12-24 hours after the injection) development of shock (anaphylactic shock) with a drop in the pulse rate and blood pressure, pallor, cyanosis, pyrexia, dyspnea, vomiting, abdominal pains, diarrhea with mucus and blood, and sometimes a general picture of collapse. These symptoms are soon followed by eruption and other symptoms of the serum sickness. The most dangerous in this respect is intravenous administration of the serum to children who are being reinoculated.

An immediate reaction does not, as a rule, occur if more than six months have elapsed since the first administration of the serum. However, the organism continues to be highly sensitive and a second administration of the serum may produce an *accelerated* reaction.

In an accelerated reaction all the phenomena occur during the first five or six days of incubation and are more strongly pronounced than usual.

Pathogenesis The serum sickness is caused by the action of a foreign (horse) protein administered not through the intestinal tract, but directly into the blood or tissues (parenterally) where the products of its disintegration evoke a peculiar reaction of the whole organism. The essence of the reaction consists in the changes which the whole organism undergoes. The metabolism (water, protein, carbohydrate and gas) is considerably altered and the reactions of the nervous system, cardiovascular apparatus and bone marrow are modified.

Anaphylaxis is a Greek term meaning "without defence".

The *incidence* of the serum sickness caused by administration of various therapeutic serums greatly varies (10-70 per cent, averaging about 25-50 per cent) and depends on the method of preparation of the serum, its dose and method of administration, on whether or not Besredka's method was used for purposes of desensibilisation, whether or not the serum is administered for the first time or is being readministered, and, lastly, on the individual properties of the organism.

The serum sickness is far from immaterial to the organism. *In addition to the unpleasant manifestations in the form of eruption, itching and pain in the joints produced by it*, it is characterised by a certain *cardiac weakness* which is very dangerous for the patient who may be affected with diphtheritic myocarditis; moreover, the serum sickness may contribute to aggravation of the basic process and increase the organism's susceptibility to other infectious diseases. Thus it is not infrequently attended with aggravation of purulent processes, aggravation of pneumonia, inflammation of the kidneys, reappearance of a false membrane on the fauces or aggravation of the manifestations in croup,* and aggravation of dysentery.

Lastly, the serum sickness may facilitate infection with intra-hospital diseases brought into hospital departments. Because of all the aforesaid it is necessary carefully to consider the indications for administration of the serum, especially its repeated administration.

To prevent anaphylactic phenomena in cases of repeated serum administration, there must be no intervals exceeding six or seven days.

During administration of serum it is necessary very strictly to observe the rules of asepsis so as to avoid introduction of infection; the serum should preferably be administered into the thigh (not a buttock or the abdomen).

Prophylaxis. The most efficient method is freeing the therapeutic serums of the superfluous protein,** i.e., production of so-called

* These aggravations of the inflammatory process on the mucous membranes with appearance of exudate and aggravation of stenosis are not a *relapse* of diphtheria, as was formerly believed, but often an *anaphylactic* process, for which reason no new portions of serum must in such cases be administered. In cases of croup repeated intubations must be avoided during this period and may be resorted to only in emergencies.

** Latterly an antidiphtheric serum subjected to dialysis and the action of the enzymes of the gastric juice (a so-called "diaferm" serum, i.e., dialysed and fermented) has been recommended for the purpose of decreasing the frequency and severity of the serum sickness. Administration of this serum reduces the incidence of the serum sickness and eliminates its severe forms.

dialysed, purified serums which yield a lower serum sickness incidence and, what is even more important, render its course milder.

In administering the usual serums it is important that the intervals between the injections should not exceed seven days. It is also necessary carefully to collect the anamnesis of children who were ever given a serum injection and avoid administering intravenous injections for fear of producing anaphylactic shock. Sensitivity to serum is retained for seven years after administration of any serum.

The sensitivity of the organism is highest during the first 3-5 months after the injection.

In cases of usual intramuscular administration Besredka's method must *always* be employed, i.e., one and a half to two hours before administration of the bulk of the serum it is necessary to inject 1 ml of it into the muscle. This method prevents anaphylactic phenomena and moderates the severity of the serum sickness symptoms. The serum must be *well heated* and administered slowly. During the serum sickness administration of the serum into the *vein and spinal canal* has to be temporarily *suspended*.

During this period 0.5-1 ml of (1:1,000) adrenalin is added to the serum being administered subcutaneously or intramuscularly.

Treatment. In cases of *itching*—sponging the skin with water containing alcohol or vinegar. Warm baths produce a sedative effect. For *pains in the joints*—heat and a splint. For *shock*—subcutaneous administration of 0.5-1 ml of (1:1,000) adrenalin and other cardiacs—ephedrine, caffeine, cordiazol and cordiamine.

Administration of *insulin* (5-10 u) twice a day simultaneously with glucose, as well as *endonasal administration of adiurecrine* (desiccated preparation of the posterior pituitary), is also helpful. The latter is administered in a dose of 0.025 g once or twice a day.

Recently obtained data attest good therapeutic effect of dimedrol (diphenhydramine) administered in doses of 0.01-0.03 g (depending on age) two or three times a day.

1. Agents Used in Respiratory Diseases
(Expectorants, Antitussives, Stimulants of the Respiratory Centre)

Age Preparation	Up to 1 year	1-3 years	3-5 years	5-7 years	7-10 years	10-12 years	12-14 years	Method of administration
Ipecacuanæ radix (Ipecac)	not to be prescribed	0.01	0.01	0.015	0.015	0.02	0.025	In an infusion of 0.1-0.2 per 100 ml, a tea-, dessert- or tablespoonful 4-6 times a day (expectorant)
Natr. bicarbonicum (Sodium bicarbo- nate)	0.1-0.2	0.3	0.3	0.5	1.0	1.5	1.5	In a 2-3 per cent solution, a tea-, dessert- or tablespoon- ful with an addition of 1.0-2.0 Liq. Ammonii ani- sati (expectorant)
Liquor Ammonii anisati	1 drop	3 drops	3 drops	5 drops	8 drops	10 drops	10 drops	As a 1-2 per cent solution added to expectorant mixtures (expectorant)
Pertussin	10-20 drops	1-2 tea- spoonful	1 tea- spoonful	1 tea- spoonful	1 de-sert- spoonful	1 table- spoonful	1 table- spoonful	3-4 times a day (administered for whooping cough)
Therpinum hydra- tum (Terpin hyd- rate)	0.03-0.05	0.05-0.1	0.1	0.15	0.2	0.25	0.3	2-3 times a day (expectorant and disinfectant)

(Continued)

A _{ae}		Up to 1 year							Method of administration						
Preparation		1 3 years	3 5 years	5 7 years	7 10 years	10 12 years	12 14 years								
Pulvis Doveri (Dover's powder) (mixture of opium, ipecac and milk sugar)	not to be pre-crilled	0 03	0 05	0 1	0 15	0 2	0 25	2 3 times a day (antitussive and partly expectorant)							
Codeinum phosphoricum (Codeine phosphate)	ditto	Number of ml to equal the number of years in the child's age													
Codeinum purum (Pure codeine)	ditto	Half the dose of codeine phosphate							2 3 times a day in powders (antitussive)						
Dioninum (Dionin)	ditto	Number of ml to equal the number of years in the child's age													
Cytitonum (cytisine) (0.15 per cent aqueous solution of cytisine)	0 10 15 ml	0 2-0 25 ml	0 3 ml	0 5 ml	0 75 ml	1 ml	1 ml	Used for stimulating the respiratory centre in asphyxia of newborn, shock, weak respiration and lowered blood pressure, in infectious diseases (pneumonia, dysentery, typhus, etc.)							
Lobelinum (lobeline) (1 per cent solution of hydrochloride of lobeline)	0 1-0 25 ml	0 3 ml	0 4 ml	0 5 ml	0 75 ml	1 ml	1 ml								

2. Cardiovascular Stimulants and Tonics

Preparation	Age	Up to 1 year	1-3 years	3-5 years	5-7 years	7-10 years	10-12 years	12-14 years	Method of administration
Adonisid vernalis herba (Adoniside)		not to be prescribed	0.15	0.2	0.25	0.3	0.4	0.5	As an infusion in a dose of 2.0-2.5:100.0; a tea-, dessert- or tablespoonful; often Bekhterev's mixture or tablets together with sodium bromide and codeine
Adoniten (Adonilene)		ditto	The number of drops per dose to equal the number of years in the child's age						2 or 3 times per day
Coffeinum natrio-benzolicum (Caffeine and sodium benzoate)		0.02-0.05	0.05	0.08	0.1	0.1	0.15	0.2	In powders or a 0.5-1 per cent solution, a tea-, dessert- or tablespoonful 3 or 4 times per day
Coffeinum natriosalicylicum (Caffeine with sodium salicylate)									
Coffeinum purum (Pure caffeine)			Half the preceding dose						In powders 3 or 4 times per day, or subcutaneously

(Continued)

Preparation	Age	Up to 1 year	1-3 year	3-5 year	5-7 year	7-10 years	10-12 years	12-14 year	Method of administration
Coffeinum natrio benzolicum (Caffeine and sodium benzoate) (10 per cent solution for injections in ampoules)		0.3 ml	0.5 ml	0.7 ml	0.7 ml	0.8 ml	1.0 ml	1.0 ml	Subcutaneously 2 or 3 times per day
Cordiaminum (Cordiamine) (for injections)		1-2 drops	2-4 drops	3-5 drops	5-7 drops	7-10 drops	10-12 drops	12-14 drops	Per os 2 or 3 times per day
Cordiaminum (Cordiamine) (for injections)		0.1-0.15 ml	0.2 ml	0.3 ml	0.4 ml	0.5 ml	0.6 ml	0.75 ml	Subcutaneously 2 or 3 times per day
Digitalis tolum (Powdered digitalis)		0.005	0.01	0.02	0.03	0.05	0.06	0.075	In powders 3-5 times per day or a water infusion 0.1-0.3 100, or tea, dessert or table-spoonful
Digitalinum (Digitalinum) (Digitalin)		The number of drops per dose to equal the number of year in the child's age							
Digalen neo (purified Digitalis feruginea extract for injections)		0.1-0.2 ml	0.3 ml	0.5 ml	0.6 ml	0.75 ml	1.0 ml	1.1 ml	Subcutaneously 1 or 2 times per day

Oleum camphoratum (Camphor oil) (10 per cent, for injections)	0.5-1.0	1.0	1.0-1.5	1.5	1.5-2.0	2.0	2.0	Subcutaneously 3 or 4 times per day
Strychninum nitricum (Strychnine nitrate) (for internal use)	not to be prescribed	0.0002	0.0003	0.0005	0.0006	0.0007	0.001	Per os; young children— in a weak solution (1:100,000), older chil- dren—in pills, 2 or 3 times per day
Strychninum nitricum (Strychnine nitrate) (in a 1:1,000 dilution)	ditto	0.1	0.2	0.3	0.4	0.5	0.5	Subcutaneously 2 or 3 times per day; the dose may be increased for diphtheria
Tinctura Strychni (Nux vomica tincture) (in drops)	ditto	1 drop	2 drops	3 drops	4 drops	5 drops	5 drops	2 or 3 times per day
Tinctura Strophanti (Strophanthus tincture) (in drops, per os)	ditto	1 drop	2 drops	3 drops	4 drops	5 drops	5 drops	2 or 3 times per day
Tinctura Convallariae majalis (Convallaria majalis tincture) per os	1-2 drops	2-3 drops	4-5 drops	5-6 drops	7-8 drops	10 drops	15 drops	2 or 3 times per day

3. Agents Used in Gastrointestinal Diseases (Laxatives, Astringents, Opiates, Disinfectants, Sedatives, Appetisers, etc.)

Preparation	Age						Method of administration
	Up to 1 year	1-3 years	3-5 years	5-7 years	7-10 years	10-12 years	12-14 years
Oleum Ricini (Castor oil)	1 tea-spoonful	1 dessert-spoonful	1 dessert-spoonful	1 table-spoonful	1 table-spoonful	1 table-spoonful	1 table-spoonful
Oleum Vaselini (Vaseline oil)	1 tea-spoonful	1 dessert-spoonful	1 dessert-spoonful	1 table-spoonful	1 table-spoonful	1 table-spoonful	1 table-spoonful
Infusum Sennae compositum (Senna infusion)	not to be prescribed	1 tea-spoonful	1 tea-spoonful	1 dessert-spoonful	1 table-spoonful	1 table-spoonful	1 table-spoonful
Natr. Sulfuricum (Sodium sulfate)	The number of grams per dose to equal the number of years in the child's age						In a 2-5 per cent solution
Magnesium sulfuricum (Magnesium sulfate) (Epsom salt)							
Pulvis liquoritiae compositum	1.0	1.5	2.0	2.5-3.0	4.0	5.0	5.0
Bismutum subnitricum (Bismuth subnitrate)	0.1	0.15	0.2	0.25	0.3	0.5	0.5
Tannabinum (Tannalbin)	0.1	0.15	0.2	0.25	0.3	0.5	0.5
							In powders 2 or 3 times per day

(Continued)

Salolium (Salol) (Phenyl salicylate)	0.1	0.15	0.2	0.25	0.3	0.3	0.3	In powders 2 or 3 times per day
Benzonaphtholium (Benzonaphthol) (Betanaphthyl benzoate)	0.1	0.15	0.2	0.25	0.3	0.3	0.3	
Urotropinum (Urotropin)	0.1	0.15	0.2	0.25	0.3	0.3	0.5	
Bolus alba (Kaolin)	1.0-2.0	3.0	3.0	5.0	5.0	10.0	10.0	In powders 2-4 times per day
Acidum hydrochloricum dilutum 10% (diluted hydrochloric acid)	1 drop	2 drops	3 drops	5 drops	8 drops	10 drops	10 drops	Ac. hydrochlorici diluti 1.0, aq. des. 100.0 a tea-, dessert- or table- spoonful 3 or 4 times per day
Pepsinum (Pepsin)	0.05-0.1	0.15	0.2	0.25	0.3	0.5	0.5	In powders, 3 or 4 times per day, followed by diluted hydrochloric acid
Pancreatinum (Pancreatin)	0.1-0.15	0.2	0.25	0.3	0.4	0.5	0.5	3 or 4 times per day to- gether with calcium carbonates

(Continued)

Age		Preparation						Method of administration				
		Up to 1 year	1-3 years	3-5 years	5-7 years	7-10 years	10-12 years	12-14 years				
Calcium carbonicum (Calcium carbonate)		0 1-0 2	0 2	0 3	0 5	0 5	1 0	1 0	3 or 4 times per day together with pancreatin			
Carbolenum (Carbon)		1 0 2 0	3 0	3 0	5 0	5 0	10 0	10 0	For abdominal pain in meteorism, 2 or 3 times per day			
Extractum Belladonae (Belladonna extract)		The number of milligrams per dose to equal the number of years in the child's age							In powders, 2 or 3 times per day			
Papaverinum hydrochloricum (Papaverine hydrochloride)		0 0 2-0 005	0 0 5-0 01	0 01	0 015	0 0 2	0 0 25	0 03	Prescribed as antispasmodic 2 or 3 times per day			

4. Vermifuges

Preparation	Age	Up to 1 year	1-3 years	3-5 years	5-7 years	7-10 years	10-12 years	12-14 years	Method of administration
Extractum Filicis maris aethereum Ether extract of filicis maris		not to be prescribed		0.5 for each year of life, but not more than 5.0. To expel the dwarf tapeworm the dose is reduced to 0.15 for each year of life					To be taken within 1-1.5 hours, dividing the indicated amount into 3 or 4 doses and taking them at 15-20-minute intervals. To expel the dwarf tapeworm the treatment is administered in 3 cycles at 10-12-day intervals
Osarsolum (Osarsol Acetarsonne)		ditto		To expel the whipworm the child is given as many tablets (of 0.25) per course of treatment (5-6 days) as there are years in the child's age					The daily dose is divided into 3 or 4 portions
Sancaphenum (Sancaphene)		ditto		The number of tablets per course of treatment to equal the number of years in the child's age					The course of treatment is 3 days; the daily dose is administered in 2 portions at a 30-minute interval
Santoninum (Santonin)		ditto		0.005 per dose for each year of life					2 or 3 times a day for 2 or 3 days
Semen cucurbitae (shelled pumpkin seed)		ditto	50.0	75.0	100.0	125.0	150.0	200.0	Shelled and bruised; given as a powder with sugar; the indicated dose is taken in the course of a day

(Continued)

Preparation	Age	Up to 1 year	1-3 years	3-5 years	5-7 years	7-10 years	10-12 years	12-14 years	Method of administration
Sulfur depuratum									3 times per day for 4 days; after an intermission the preparation is given for 4 more days for a total of 5 such cycles. Administered to expel pinworms
Sulfur praecipitatum (Washed sulfur) (Precipitated sulfur)		ditto	0.1-0.2	0.25	0.3	0.5	0.6	0.8	
Diuretinum (Deuretin)		0.003-0.05	0.1	0.15	0.25	0.3	0.4	0.5	In powder 3 or 4 times per day and in mixture together with cardiacs
Folium Uvae ursi (Uva ursi)		0.1-0.15	0.2	0.25	0.3	0.4	0.5	0.5	As a decoction of 3.0-5.0:100.0 a tea, dessert- or tablespoonful 3-4 times per day
Liquor Kalii acetici (Potassium acetate)		0.3	0.5	0.75	1.0	1.0	2.0	2.0	In mixtures combined with cardiacs 3 times a day
Mercusalum (Mercusal) (Mersalyl)		0.1-0.2	0.25	0.3	0.5	0.75	1.0	1.0	Intramuscularly 2 or 3 times a week

6. Sedatives (Hypnotics, Antispasmodics and Narcotics)

Preparation	Age	Up to 1 year	1-3 years	3-5 years	5-7 years	7-10 years	10-12 years	12-14 years	Method of administration
Adalinum, Bromuralum, Medinalum (Adalin, Bromural, Medinal)		0.05 0 1	0.15	0 2	0 2	0 2	0 3	0 3	In powders for the night
Luminalum (Luminal)		0 01	0 02	0 03	0 03	0 05	0 1	0 1	Given as a hypnotic for the night, sleep setting in 1-1.5 hours. In cases of migraine the dose for older children is reduced to 0.03 and is combined with pyramidon (0.2) and caffeine (0.05)
Chloralum hydratum (Chloral hydrate) (2 per cent solution for enema)		10 0 15 0 15	0-20 0 15	0 20 0 30 0 40 0	0 30 0 40 0	40 0	40 0	50 0	A chloral hydrate enema is administered after a cleansing enema
Kalum bromatum (Potassium bromide)		0 05	0 1	0 15	0 2	0 25	0 3	0 5	As a 0.5-1.2 per cent solution; a tea-, dessert- or tablespoonful 2 or 3 times per day. Bromide preparations may be combined with caffeine—0.25 per
Natrium bromatum (Sodium bromide)									

(Continued)

Preparation	Age						Method of administration
	Up to 1 year	3 years	3-5 years	5-7 years	7-10 years	11-15 years	
Calcium bromatum (Calcium bromide)	0.1	0.2	0.3	0.4	0.5	0.6	cent Sodium bromide and 100.0 Caffeine and Sodium benzoate 0.1-0.2, a tea, dessert- or tablespoonful 4 times per day
Calcium chloratum (Calcium chloride)	0.25-0.5	0.5	0.75	1.0	1.0	1.5	As an antispasmodic in a 10 per cent solution, a tea-, dessert- or tablespoonful 4-6 times a day
Calcium gluconicum (Calcium gluconate)	0.3-0.5	0.5	1.0	1.5	2.0	2.5	In powders 3-5 times per day
Tinctura Valerianae (Valerian tincture)	2 drops	5 drops	5 drops	8 drops	10 drops	15 drops	2 or 3 times per day
Valerianae radix (Valerian root)	0.1	0.15	0.2	0.3	0.4	0.5	In a 2-3-0-100 infusion, usu- ally together with bromides, a tea-, dessert- or tablespoonful 3 times per day
Pantoponium (Pantopon)	not to be prescribed						In powders once or twice a day, in drops—1 per cent solution, 1 drop for each year of the child's age

7. Antipyretics, Sudorifics and Antimalarial Agents

Preparation	Age	Up to 1 year	1-3 years	3-5 years	5-7 years	7-10 years	10-12 years	12-14 years	Method of administration
Aspirinum (Aspirin)		0.05	0.1-0.15	0.2	0.25	0.3	0.4	0.5	In powders 2 or 3 times per day
Natrium salicylicum (Sodium salicylate)		0.1-0.15	0.2	0.25	0.3	0.4	0.5	0.5	2 or 3 times per day; larger doses are administered in rheumatism—0.5 per day for each year of the child's age
Phenacetinum (Phenacetin)		0.02-0.05	0.1	0.15	0.2	0.25	0.25	0.3	In powders 2 or 3 times per day, often in combination with caffeine, aspirin, pyramidon (for headache)
Pyramidonum (Pyramidon)		0.03-0.05	0.05-0.1	0.1	0.1-0.15	0.15	0.2	0.25	In powders 2 or 3 times per day (for headache). For rheumatism the doses are increased to 0.15-0.2 a day for each year of the child's age, but not more than 1.5-2.0 per day
Acricinium (Acricine) (Quinacrine)		0.008	0.01-0.02	0.02-0.025	0.03-0.04	0.05	0.06	0.08	2 or 3 times per day
Ditto (4 per cent solution for injections)		0.5-1.0	1.0-1.5	1.5-2.5	2.5-3.0	4.0	5.0	6.0	Once a day intramuscularly
Chininum hydrochloricum (Quinine hydrochloride)		Number of cg per day to equal the number of months in the child's age and dg—the number of years in the child's age							
Euchininum (Euchinine)		50 per cent more than quinine							
		Taken in 3 or 4 doses							
		In 3 or 4 doses							

8. Agents Used for Various Blood Diseases

Preparation	Age	Up to 1 year	1-3 years	3-5 years	5-7 years	7-10 years	10-15 years	15-20 years	Method of administration
Ferrum hydrogeno reducti (Reduced iron)		0.2	0.3	0.5	0.5	0.6	1	1	3 times per day followed by diluted hydrochloric acid
Ferrum carbonicum (Ferrous carbonate)		0.2	0.3	0.5	0.5	0.6	1	1	
Ferrum lacticum (Ferrous lactate)		0.2	0.3	0.5	0.5	0.6	1	1	
Ferrum glycerophosphoricum (Iron glycerophosphate)		0.1	0.2	0.25	0.3	0.4	0.5	0.5	
Liquor Ferri albuminati (Iron albuminate)		10 drops	25 drops	1-2 tea spoonful	1-2 tea spoonful	1-2 tea spoonful	1-2 tea spoonful	1-2 tea spoonful	3 times per day
Sirupus Ferri jodati (Ferrous iodide)		5-10 drops	15 drops	20 drops	20 drops	20 drops	40 drops	40 drops	
Haematogenum (Hematogen)		1/2 tea spoonful	1 tea spoonful	1 tea spoonful	1 tea spoonful	1 tea spoonful	1 tea spoonful	1 tea spoonful	

9. Sulfu Drugs and Antibiotics

Preparation	Age	Up to 1 year	1-3 years	3-5 years	5-7 years	7-10 years	10-12 years	12-14 years	Method of administration
Streptocidum album (White streptocide)		0.1	0.2	0.3	0.4	0.5	0.5	0.5	3 or 4 times per day
Albucidum (Albucide)									4 times per day; albucide is additionally used in the form of drops for the treatment of eye diseases (10-30 per cent solution, 2 drops into each eye 2 or 3 times per day)
Sulfidinum (Sulfidine)									
Sulfadimezin									
Sulfasolum (Sulfazole)		0.1-0.2	0.25	0.3	0.4	0.5	0.7	1.0	
Norsulfasolum (Norsulfazole)									
Disulfordinum (Disulformin)									
Phthalasolum (Phthalazole)									
Penicillinum (Penicillin) (for injections)		Children up to 1 year of age daily dose of 10,000-20,000-30,000 u per 1 kg of weight, depending on the severity of the disease. Older children—from 150,000 to 400,000 u per day							The daily dose is divided into 2 or 3 injections
Ecmolinum (Ecmolin) (0.5 per cent solution)		Used for prevention of influenza 2 or 3 drops being instilled into each nostril. The preparation is also added to a penicillin solution in a per cent novocain solution to prolong its action							
Biomycinum (Biomycin) Terramycinum (Terramycin)		0.025 (25,000 u) per 1 kg of weight per day							Daily dose given in 3 portions

(Continued)

Preparation	Age						Method of administration
	Up to 1 year	1-3 years	3-5 years	5-7 years	7-10 years	10-12 years	12-14 years
Tetracyclinum (Tetracycline)	0.025 per 1 kg of weight per day						Daily, dose given in 3 portions
Streptomycinum (for intramuscular injections; daily dose for the treatment of tuberculosis)	0.2	0.25	0.25	0.3	0.5	0.5	The daily dose is administered in 2 stages, 0.1 g of the preparation equalling 100,000 u
Streptomycinum (for injections into the spinal canal in the treatment of tuberculous meningitis)	0 1-0.015	0.015-0.025	0.025-0.03	0.04-0.05	0.05-0.075	0.1	Once a day
Albomycinum (Albomycin)	Administered to children in the first year of life in the treatment of pneumonia, otitis and antritis in a dose of 100,000-200,000 u per 1 kg of weight for 3 injections						Course of treatment is 7-12 days
Sintomycinum (Synthomycin)	Children weighing less than 16 kg are prescribed 0.02 g per 1 kg of weight per dose; children weighing more than 16 kg and up to 14 years of age—0.25-0.4						4 times per day
Levomycetinum (Levomycetin)	The dose is half that of synthomycin						4 times per day

10. Vitamin Doses Recommended in the Treatment of Children

Vitamin	For internal use		Daily dose for parenteral administration	Notes
	Single dose in mg	Daily dose in mg		
A	2-3	6	Not administered parenterally	
B ₁ (thiamine)	5-10	30	10	In one or several injections
B ₂ (riboflavin)	2-10	20	Not administered parenterally	
		50; in	50	In 2 or 3 injections
PP (nicotinic acid)	1-20	pellagra up to 200	in pellagra up to 200	In one or several injections
C (ascorbic acid)	100	300	50-300	
D		5,000-10,000	Not administered parenterally	
K (vikasol)	15	15	Ditto	Not to be taken for more than 3 days in succession

Approved by the Pharmacological Committee of the U.S.S.R. Ministry of Public Health, January 1, 1948 (Protocol No. 1).

11. Drops and Ointments Most Frequently Used in the Treatment of Children

Nose Drops

Rp. Sol. Adrenalini 1:1,000 1.0
2% Sol. acidi borici 10.0
DS. 2-3 drops b.i.d. or t.i.d.

Rp. Sol. Protargoli 1% 10.0
S. 2-3 drops b.i.d. or t.i.d.

Rp. Sol. Rivanoli 1:1,000 10.0
S. 2-3 drops b.i.d. or t.i.d.

Rp. Penicillini 50,000 u.
Aq. destill. 5.0
DS. 3-4 drops t.i.d. or q.i.d.

Eye Drops

Rp. Sol. Albucidi 30% 10.0
DS. 2 drops b.i.d.

Rp. Sol. Collargoli 4% 10.0
DS. 2-3 drops b.i.d.

Rp. Zinci sulfurici 0.1
Aq. destill. 10.0
DS. 2 drops b.i.d.

Ointments

Rp. Ung. Wilkinsonii
Vaselini flavi aa 30.0
MDS. Ointment for itching

Rp. Ung. zinci
Vaselini aa 10.0
Naphthalani 1.0
MDS. Ointment for chafing

Rp. Anaestesini 1.0
Naphthalani
Bismuthi subnitrici
Zinci oxydati aa 6.0
Vaselini flavi 60.0
MDS. Ointment for eczema

Rp. Zinci oxydati

Amyli aa 25.00

Aq. Plumbi sive

Glycerini

Aq. destill. aa 25 0

MDS. Thin ointment for eczema
and redness of the skin
without intense exudation

Rp. Anaesthesini

Balsami peruviani aa 0.5

Bismuthi subnitrici 8.0

Vaselin_i flavi 40.0

MDS. Ointment for nipple fissures

Rp. Ung. hydrargiri oxydati

flavi 1% 15.0

DS. Ointment for eyes and nose

Rp. Chlorali hydrati

Camphorae tritae aa 2.0

Lanolini

Vaselin_i aa 10.0

MDS. Ointment for itching skin
(urticaria in the serum
sickness)

Rp. Bryllianti gruni 0.1

Spir. Vini rectific. 70° 10.0

MDS. For painting pustules on
the skin

Rp. Gentiani violeti 0.1

Aq. destill. 10.0

MDS. For swabbing the oral mucosa in thrush

12. Most Important Forms of Poisoning in Childhood

Poison and principle (in parentheses)	Clinical Phenomena	Treatment
Benzine, kerosene (phenol)	Vomiting (with a kerosene odour) abdominal pain, at first excitement and then unconsciousness, irregular respiration, cardiac weakness	Fresh air, gastric lavage with boiled water, plenty of milk to drink, castor oil, sodium sulfate, and enemas for several days. In cases of respiratory arrest—artificial respiration. In cases of collapse—subcutaneous administration of camphor and caffeine
Mushrooms: toadstools, fly agarics (muscarine) certain species of morels	Vomiting, abdominal pain, constriction of the pupil of the eye, clouded consciousness, sometimes delirium, slow, soft pulse, laboured, irregular respiration	Gastric lavage first with potassium permanganate (1-3,000) and then water; castor oil or sodium sulfate per os, plenty of strong tea and muculent decoctions to drink. Repeated enemas for several days. In cases of collapse—camphor and caffeine subcutaneously. Sour drinks and vinegar prohibited
Scopolamine, henbane (hyoscyamine), belladonna (atropine)	Dryness and burning in the mouth, intense thirst, dilation of the pupils, flushed face, vomiting, sometimes diarrhea with blood, great excitement and hallucinations, sometimes cardiac weakness	Gastric lavage, castor oil or sodium sulfate per os, repeated enemas, in cases of excitement—chloral hydrate in the enema, 4 per cent sodium bromide solution—1 teaspoonful (desert spoonful) 4-6 times per day (per os). In cases of collapse—camphor subcutaneously
Acids (acetic, carbolic, etc.)	Burns and pains in the oral cavity, fauces, stomach and abdomen. Vomiting brown-yellow or bloody masses. Characteristic odour from	Gastric lavage with water only immediately after the poisoning; plenty of water to drink. Emetics and peroral administration of carbon-

Poison and principle (in parentheses)	Clinical Phenomena	Treatment
	<p>the mouth in cases of poisoning with carbolic and acetic acid General weakness, pallor, dyspnea. Weak, rapid pulse Pulmonary edema and collapse are possible Scant urination Urine contains albumin and blood</p>	<p>alkalis are contraindicated for fear of rupturing the gastric walls by the forming carbon dioxide. Per os—calcined magnesia (20.0-200.0) lime milk egg white muculent decoctions The burned mucosa of the mouth and pharynx should be painted with a 2 per cent cocaine or dicaine solution In cases of collapse caffeine and camphor, Ringer's solution or a 5 per cent glucose solution subcutaneously, 10 ml of a 10 per cent calcium chloride solution intravenously. Pieces of ice to be swallowed</p> <p>The limbs above the point of the bite must be quickly and tightly bandaged and the wound cauterised with red-hot iron or a 10 per cent iodine tincture Wet cups at the point of the bite 2 or 3 ml of a 4 per cent potassium permanganate or hydrogen peroxide solution subcutaneously close to the point of the bite. If not more than 4 hours have elapsed since the bite—subcutaneous injection of antiscorbutic serum, caffeine, camphor and physiologic saline solution. Plenty of hot strong tea or coffee to drink</p>
Snake poison (snakebite)	<p>Swelling and redness at the point of the bite Swelling of the glands and formation of blisters at the point of the bite. Dizziness, vomiting, diarrhea Cold skin Small and rapid pulse, dyspnea, collapse</p>	
Santonin, santonica seed	<p>Everything seems yellow-coloured, the pupils are dilated, headache, vomiting, diarrhea,</p>	<p>Gastric lavage, sodium sulfate per os; repeated enemas. In cases of convulsions—chloro-</p>

(Continued)

Poison and principle (in parentheses)	Clinical Phenomena	Treatment
Fowler's solution, osarsol, neosalvarsan (arsenic)	yellow urine, sometimes cardiac weakness and convulsions Intense pain in the throat, esophagus and stomach, thirst, vomiting, sometimes with blood, intense choleralike diarrhea resembling rice water, urinary retention, weakness, pallor, feeble pulse. Paralysis of limbs may result	oral hydrate in enemas, warm baths. Plenty of tea and water to drink. In cases of collapse—camphor and caffeine subcutaneously. Milk, all fats and castor oil to be avoided Gastric lavage with calcined magnesia—20.0 per 1 litre of warm water; arsenic antidote—1 teaspoonful (dessertspoonful) every 10 minutes. Plenty of milk to drink; sodium sulfate per os. In cases of collapse—camphor and caffeine subcutaneously. In cases of poisoning after neosalvarsan—1/4-1/2 of a 1-g syringe of adrenalin (1:1,000) subcutaneously or intravenously. Acid and alkaline drinks must be avoided
Narcotics: morphine, opium, pantopon, codeine	Constriction of pupils, dizziness, intense somnolence developing into sleep, slow respiration, weak pulse	Repeated gastric lavage, first with a potassium permanganate solution (1:3 000) and then water, plenty of strong tea and coffee to drink. The child must not be allowed to sleep. Hot baths with cold dousing. Repeated enemas. Sodium sulfate per os, when the child has regained consciousness. Camphor and caffeine subcutaneously
Turpentine	Vomiting pain in the stomach, dizziness, characteristic odour from the mouth, intense excretion of urine (urine smells of violets) Protein and blood in urine. Cardiac failure	Gastric lavage; sodium sulfate per os; repeated enemas, plenty to drink, muculent soups. In cases of collapse—camphor and caffeine subcutaneously

Poison and principle (in parentheses)	Clinical Phenomena	Treatment
Mercuric chloride and calomel	Metallic taste in the mouth. Grey film on the lips and tongue, salivation, pain on swallowing, abdominal pain, bloody vomit, mucosanguineous feces. Retention or absence of urine. Slow and feeble pulse. Cold sweat. If the child survives, severe stomatitis and nephritis often develop.	During the first 3 hours after the poisoning—gastric lavage (cautious) with a calcined magnesia solution (20.0 per 300 ml of water). Later lavage is contraindicated because of the danger of perforating the esophagus and stomach. Castor oil and calcined magnesia per os. Plenty to drink—milk with scrambled egg white. Repeated warm enemata. In cases of collapse—camphor and caffeine subcutaneously. Common salt and hydrochloric acid solutions must be avoided.
Phosphorus, phosphoric matches	Vomiting, abdominal pain, specific odour of the exhaled air; the vomit not infrequently shines in the dark; rapid, feeble pulse, icterus, enlarged liver.	Numerous gastric lavages. Water and muculent soups in large quantities; sodium sulfate per os. Repeated enemata. In cases of cardiac failure—caffeine and camphor subcutaneously. Milk, fats and castor oil to be avoided.
Food poisoning	Headache, abdominal cramps, vomiting, diarrhea, dilatation of the pupils, weakness, cardiac weakness	Numerous gastric lavages with a potassium permanganate solution (1:3,000) and then water. Emetic (0.5 per cent apomorphine) subcutaneously; sodium sulfate per os, heat to abdomen, plenty of strong tea to drink, muculent soups during the first 2-3 days. Antibotulinus serum intravenously. In cases of collapse—caffeine and camphor subcutaneously; subcutaneous infusion of Ringer's solution and a 5 per cent glucose solution
Alkalis (caustic soda, caustic potash, ammonia, ammonia water)	Burns of the lips, oral esophageal and gastric mucosa. Characteristic odour in cases of ammonia water poisoning. General weakness,	Earliest possible cautious gastric lavage with water acidified with acetic acid (the tube must be well-greased). Per os—repeatedly,

(Continued)

• Poison and principle (in parentheses)	Clinical Phenomena	Treatment
	pallor, irregular respiration. Feeble, quick pulse	a 1 per cent acid solution (acetic, citric, tartaric) in tablespoonfuls every 5 minutes. Muculent decoctions containing the same acids. Plenty of milk to drink. Bed rest. In cases of alkaline skin affection the skin must be washed with water and fomentations with the same acids (5 per cent solutions) applied to the burned points. Ringer's solution or physiological saline solution and a 5 per cent glucose solution subcutaneously. In cases of intense pains—morphine or pantopon

INSTRUCTIONS FOR COMPULSORY SMALLPOX VACCINATION

1. All citizens of the U.S.S.R. are subject to compulsory smallpox vaccination.

Primary vaccination is administered at the age of 5-8 months and for epidemic indications earlier.

Note. In cases in which the newborn has been inoculated against smallpox to prevent the spread of the disease inoculation with the PCG vaccine is postponed for four months.

Revaccination is administered at the ages of 4-5, 10-11 and 18-20 years.

2. All persons to be vaccinated against smallpox are subject to preliminary medical examination for the purpose of establishing clinical indications against the vaccination.

The clinical indications against smallpox vaccination are:

a) acute infectious diseases, including the period of convalescence of at least two months after the disappearance of all clinical phenomena;

b) fever;

c) tuberculosis in the period of acute symptoms (high and subfebrile temperature, aggravation of the process, pleurisy, pneumonia, tuberculosis at the generalisation stage) and marked tuberculous intoxication (children up to two years of age with a positive Pirquet reaction);

(*Note.* In all doubtful cases the question of attempting immunisation is settled by consultation with a specialist in tuberculosis.)

d) acute intestinal disorders;

e) diabetes mellitus in children;

f) acute nephroso-nephritis;

g) heart diseases in the period of decompensation;

h) allergic states: asthma, rheumatism during the aggravation period;

i) marked hemorrhagic diathesis (hemophilia);

j) extensive exudative eczema and suppurative skin diseases.

Persons with the aforementioned contraindications are exempt from smallpox vaccination until disappearance of the contraindications, which is established by repeated medical examination.

Note. There are no medical indications against vaccination when the disease threatens to spread, in which cases all citizens are vaccinated.

3. Inoculation against smallpox is performed with a smallpox vaccine (dry and liquid).

Before vaccination the person to be vaccinated must bathe and put on clean underwear. Children's fingernails must be cut short.

The skin at the point of vaccination is swabbed with a cotton soaked in 70° alcohol or ether. If the skin at the point of vaccination happens to be soiled, it must be washed with soap and water, wiped and swabbed with alcohol or ether.

The use of iodine, carbolic acid and other disinfectants to disinfect the skin is prohibited.

The vaccination is administered on the lateral surface of the upper arm just below the shoulder joint.

After the alcohol has dried, 3 drops of the vaccine are applied with a vaccinating instrument to the cleaned surface of the skin at least 2 cm apart, the drops arranged in the shape of an equilateral triangle and connected by single linear nonbleeding abrasions 0.5-1 cm long.

Vaccination with double or cruciform abrasions is prohibited.

The surface of the vaccinated skin remains uncovered for 5-10 minutes.

The vaccine must not be rubbed in.

Note. If the vaccination is made with a strong vaccine, the physician responsible for the vaccinations may allow it to be performed with two abrasions, each 3 mm long.

The vaccinating instrument (vaccination needle) is thoroughly disinfected by boiling after each vaccination.

4. The results of vaccination are checked on the 7th or 8th day after the inoculation. The vaccination is considered effective if there is at least one properly developed pustule—pock.

If the results of the vaccination are negative the child must be revaccinated, usually on the day of examination, on the other arm.

If the vaccination has failed for the second time, it must be repeated within 3-6 months. The results of revaccination are checked on the 4th or 5th day after revaccination. Revaccination is considered successful if a clearly marked papule or vesicle can be observed during the check-up.

The pocks must be carefully guarded against injury. Moistening of the pocks must be avoided when bathing the child.

In case of any complication or injury to the pocks the vaccinated child must be shown to a physician.

5. The vaccine is dispensed either glycerinated (liquid) or dry (dissolved in glycerin before use).

Before application the end of the ampule containing the dry vaccine is wiped with sterile cotton and cut with a special emery wheel, following which the end of the ampule is broken off.

The solvent—50 per cent sterile glycerin—is introduced into the aforesaid ampule from another ampule by means of a sterile dropper; both the dropper and ampule with glycerin are dispensed together with the vaccine.

0.2 ml (6 drops) of the solvent is introduced into a 20-dose ampule and 0.1 ml (3 drops) into a 10-dose ampule, the contents of the latter being thoroughly stirred with the end of the vaccination needle or a sterilised glass rod.

In an open ampule the smallpox vaccine can be used only in the course of one day, after which it is destroyed.

Vaccination may be administered only by appropriately trained medical people.

6. The dry smallpox vaccine must be stored in a dark, cool place at a temperature not above 10°C, the liquid vaccine—not above 6°C.

The vaccine-containing ampule must have a label indicating the institution in which the vaccine was prepared, the number of doses, the number of the series, date, period of validity and state control number.

A vaccine in a cracked ampule or improperly labelled, as well as unlabelled, is not fit for use.

The institution in which the vaccine was prepared should be addressed for all information concerning administration of the vaccine.

The period of validity of the vaccine must be indicated on the label.

Approved by the Chief Sanitary and Anti-epidemic Administration of the U.S.S.R.
Ministry of Public Health, May 20, 1953

**INSTRUCTIONS FOR COMPULSORY INOCULATIONS
AGAINST DIPHTHERIA
(ACTIVE IMMUNISATION WITH DIPHTHERIC ANATOXIN)**

1. Children from the end of the first year of life (11-12 months) up to 12 years of age are subject to compulsory inoculation against diphtheria.

Children up to eight years of age are the first to be inoculated. Persons 13 years old and older are inoculated only on epidemic indications in the given locality.

Epidemic indications for inoculation of these age groups are a high diphtheria incidence among these groups, increased diphtheria incidence in the given locality and an increasing share of these groups in the general morbidity.

Inoculations for epidemic indications must be performed only by agreement with the regional or city public health departments.

In cases of outbreaks of diphtheria in children's institutions, tenements, etc., especially epidemic outbreaks of the disease, it is necessary, for epidemic indications, to check up on the immunity of all children and immediately to inoculate the formerly uninoculated children.

2. All persons subject to inoculation must undergo a preliminary medical examination for clinical indications against inoculation, necessarily including thermometry directly before inoculation.

Clinical indications against inoculation are:

a) acute infectious diseases, including the period of convalescence of at least two months after disappearance of all clinical manifestations;

b) fever;

c) tuberculosis in the period of acute manifestations (subfebrile temperature, aggravation of the process, pleurisy, pneumonia, tuberculosis in the generalisation stage) and during its chronic course with flare-ups, marked intoxication, and a positive Pirquet reaction in children up to two years of age;

Note. In all doubtful cases the question of immunising children is settled by consultation with a specialist in tuberculosis.

d) acute intestinal disorders;

e) blood diseases (malignant or strongly pronounced anemia), leukemia, hemophilia;

f) diabetes mellitus;

g) acute nephroso-nephritis and periodically aggravated chronic processes, pyuria;

h) heart diseases in the period of compensation and subcompensation;

i) allergic states: rheumatism, including chorea, asthma, food and other idiosyncrasies contained in the anamnesis and confirmed by the physician;

j) spasmophilia.

Note. Children who have survived infectious diseases of the central nervous system (encephalitis, meningitis, poliomyelitis) are inoculated in each individual case after consultation with specialists.

The indications against inoculation of very young children (up to three years of age) include, in addition to the foregoing diseases:

- a) acute dyspeptic disorders;
- b) 2nd and 3rd degree hypotrophy;
- c) exudative diathesis with clearly marked manifestations (exudative eczema).

3. Children temporarily exempt from inoculation for medical contraindications must be kept under special observation and inoculated upon disappearance of the contraindications.

4. Inoculations against diphtheria are made without a preliminary Schick test.

5. Inoculations against diphtheria are made with a diphtheric anatoxin containing at least 20 antigenic or immunising anatoxin units per 1 ml (1 cc).

Note. The anatoxin now manufactured in the U.S.S.R. contains at least 25 immunising or antigenic units per 1 ml (1 cc).

6. The immunisation with anatoxin consists of two vaccinations, a primary revaccination and 3 repeated revaccinations.

7. The vaccination is administered as follows: first inoculation—anatoxin dose of 1 ml followed by an interval of 20-30 days; second inoculation—anatoxin dose of 2 ml; the interval between the first and second inoculations must not be shortened, but in individual cases of a forced interruption the interval may be prolonged to 45 days. At least 60 antigenic units must be administered during the vaccination.

8. The primary revaccination consists in a single inoculation with 1 ml of the anatoxin 3-6 months after the vaccination. If, for some reason or other, the child was not revaccinated in due time, the revaccination is also performed with one injection regardless of the time that has elapsed since vaccination. It should be remembered that vaccination without subsequent revaccination does not confer adequate immunity, for which reason extension of the interval between the vaccination and the primary revaccination is allowed only in individual cases in which the child could not, for good reasons, have been revaccinated in due time.

9. Repeated revaccinations are administered at the ages of 3-4, 7-8 and 11-12 years, i.e., at intervals of from two to five years, with one inoculation of 1 ml of the anatoxin.

10. Children of any age (up to 12 years), for some reason uninoculated, are immunised according to the usual scheme (two vaccinations and subsequent revaccinations) at intervals of at least two years. In such cases the children may not receive four revaccinations.

In individual cases, if a necessity of revaccination arises in connection with epidemic indications, a shortening of the interval to not less than one year between revaccinations is allowed, provided this revaccination is taken into account and the revaccinations are subsequently administered according to the scheme. The administration of such revaccinations for epidemic indications must be approved each time by the regional or city public health departments.

11. The following changes are envisaged in the aforesaid usual scheme of inoculations:

a) in cases of a strong reaction to the first inoculation the second inoculation is administered within 20-30 days with the same dose of anatoxin (1 ml);

b) children who have survived an attack of diphtheria are, in virtue of the inconstancy of their immunity, subjected to immunisation with one inoculation of 1 ml of anatoxin not sooner than six months after the attack of the disease; subsequently they are revaccinated according to these Instructions at the same intervals and with the same doses and consideration of their age;

c) persons from nine years of age and up are vaccinated, if they were never inoculated before, with smaller doses—0.5 ml for the first inoculation and 1 ml for the second; they are revaccinated with the usual dose of 1 ml.

Note. In cases in which the Schick test is properly conducted and specially trained personnel, precisely graduated syringes and good needles are available, persons past 13 years of age may be subjected to primary immunisation by the indications of the Schick test.

12. In cases of quarantine occasioned by any infection, besides diphtheria, in a children's institution, school, tenement or hostel, the children who were not affected with the given infection are not inoculated, each individual case being agreed upon with the epidemiologist.

13. Passive immunisation of children (administration of antidiphtheric serum) may be resorted to only in exceptional cases, in which it is impossible to ensure proper medical observation of those who had contact with diphtheria patients (communities far removed from medical services).

In such cases, simultaneously with administration of the serum (3,000-5,000 au) all children must, without fail, be actively immunised with anatoxin in accordance with these Instructions.

14. Anatoxin must be stored in a dark cool place at a temperature of from 3 to 10°C. Under these storage conditions the anatoxin can be used for three years after its manufacture whose date must be indicated on the label. Anatoxin which has suffered freezing is not fit for use.

Each ampule of anatoxin and each package containing the ampules must have a label with the following information:

- a) name and address of the institution in which the preparation was made;
- b) name of the preparation and directions for use;
- c) amount (in ml or cc) of the preparation in the ampule;
- d) number of immunising units per 1 ml of the anatoxin;
- e) number of the series;
- f) number and control date of the State Control Institute;
- g) date the preparation was made (ampuled);
- h) period of validity of the preparation.

15. Anatoxin is injected according to the following rules:

Each ampule is inspected before it is opened to reveal defective (cracked) ampules; ampules fit for use are wiped with alcohol. The neck of each ampule is first wiped with alcohol or passed over a flame and removed after it is cut with a file or edge of glass. Open ampules are covered with cotton soaked in alcohol or a sterile towel and are immediately used (open ampules may not be transported from one apartment to another, one institution to another, etc.). The anatoxin is drawn into the syringe directly from the ampule.

Before use needles are placed in a steriliser with boiling water and are boiled.

The syringe must be boiled without fail for at least 30 minutes before the beginning of the inoculations.

16. All persons to be inoculated should be advised to take a bath and change their underwear before inoculations.

The skin at the point of the injection is swabbed with cotton soaked in 70° alcohol. If the skin is soiled at the point of administration of anatoxin, it must first be cleaned with benzine. After disinfection a fold of the skin is grasped with the fingers of the left hand and the needle is introduced into the subcutaneous tissue in a downward direction at the base of the fold. One or two drops of the content of the syringe are passed through each needle before inoculation.

Anatoxin is administered under the inferior angle of the scapula; this region has very few nerves and loose subcutaneous tissue, for which reason it is a good place to inject the anatoxin. Anatoxin must be administered strictly subcutaneously. It must under no circumstances be injected into the skin itself or the muscles. After the injection the point of injection is painted with iodine.

17. Inoculations with anatoxin may be accompanied by a local reaction (redness, edema and pain at the point of injection) and a general reaction (fever, general indisposition). The reactions observed usually disappear within 24-48 hours.

The reaction is considered weak if there is a 4×4-cm infiltrate and the temperature is not above 37.5°C.

The reaction is considered average if there is a 4×4-10×10-cm infiltrate without a rise in temperature or a smaller infiltrate and the temperature is above 37.5°C.

During inoculations it is particularly necessary to consider strong reactions to the injections of anatoxin accompanied by:

- a) formation of a 10×10-cm and larger infiltrate at the point of the injection;
- b) a rise in temperature to 38.6°C and higher, regardless of the local reaction;

- c) development of marked symptoms of general indisposition, regardless of the local reaction; in cases of "strong reaction" the subsequent inoculations are made with smaller doses of anatoxin as indicated in § 11-a.

18. Considering the phenomena of shock infrequently observed in particularly sensitive subjects following administration of various vaccines, it is necessary to ensure medical observation of those inoculated with anatoxin for half an hour after the injection. In cases of shock a subcutaneous injection of 0.3-1 ml of adrenalin (1:1,000), depending on age, is recommended.

19. Inoculations against diphtheria may be administered, in addition to physicians, by intermediate medical personnel, who have a secondary medical education, under general guidance and control of a physician.

20. All antidiphtheric inoculations administered to the child are recorded in the child's case history with indications of the dates of inoculation, doses and series of anatoxin, and the reactions to the inoculations.

Periods for Administering Preventive Inoculations to Children (Addendum No. 1 to Order No. 273 of the U.S.S.R. Ministry of Public Health of July 9, 1956)

As an amendment to Order No. 743 of the U.S.S.R. Ministry of Public Health of September 10, 1955, the following periods for administering prophylactic inoculations to children are established:

1) the first BCG vaccination is to be administered to the newborn in maternity homes, maternity departments of hospitals (urban and rural), collective farm maternity homes and at the newborn's homes;

2) vaccination against diphtheria is to be administered from the 5th or 6th month of life;

3) vaccination against smallpox is to be administered from the 9th or 10th month of life, or at earlier dates for epidemic indications (epidemic smallpox in remote region).

4) children of the following ages are to be revaccinated:

Type of inoculation	First inoculation	Second inoculation	Third inoculation	Fourth inoculation	Fifth inoculation
BCG vaccine	at 2 years	at 7 years	in fourth grades	in seventh grades	in 10th grades
Against smallpox	at 4 years	at 8 years	at 12 years	at 18 years	
Antidiphtheritic	at 1 year	at 3 years	at 7 years	at 12 years	

Note. Vaccination and revaccination against whooping cough are to be administered simultaneously with antidiphtheric inoculations from 1957 on.

Children prophylactically inoculated against one infection may be inoculated against another infection after a lapse of two months.

If need be, children past three years of age may be simultaneously revaccinated against two of the following infections: smallpox and diphtheria, smallpox and intestinal infections.

After attacks of acute diseases inoculations must be administered on instructions of the attending physician, but not sooner than one month after clinical cure;

5) immunisation against measles is to be administered between three months and four years of age (primarily at two years of age) to children who have never suffered from this disease but have been in contact with measles patients. Children past four years of age are to be immunised in accordance with medical indications.

